

ACUTE CHANGES IN STRENGTH AND ENDURANCE PERFORMANCE  
AND SERUM HORMONES TO SINGLE SESSION COMBINED  
ENDURANCE AND STRENGTH LOADINGS:

ORDER EFFECT IN FEMALE AND MALE ENDURANCE RUNNERS

Moritz Schumann

Master Thesis in  
Science of Sport Coaching and Fitness Testing  
Spring 2011  
Department of Biology of physical activity  
University of Jyväskylä  
Supervisor: Prof. Keijo Häkkinen

## ABSTRACT

**Schumann, Moritz 2011. Acute changes in strength and endurance performance and serum hormones to single session combined endurance and strength loadings: Order effect in female and male endurance runners. Department of Biology of Physical Activity, University of Jyväskylä, Master's thesis in Science of Sport Coaching and Fitness Testing, 76pp.**

Endurance and strength loadings are often performed concurrently by both elite and recreational athletes. The question of whether the order of exercise yields acute differences in force production and endocrine responses when both types of exercise are combined in a single session has, however, received only limited scientific attention. The purpose of this study was to examine acute changes and recovery in endurance and strength performance and serum hormone concentrations to single session combined endurance (E) and strength (S) loadings by switching the order of exercises in men and women.

A group of 10 female (34±8 years) and 12 male (38±8 years) recreationally endurance trained subjects participated in the study. All subjects took part in two loading sessions; one with E loading followed immediately by S loading (E+S) and one with the opposite order (S+E). Prior to the measurements subjects were tested for their E ( $VO_{2max}$ ) and S performance (maximal bilateral isometric leg extension,  $MVC_{max}$ ). The subjects then performed both loadings in a randomized order. S (45min) primarily focused on leg extensor muscles including both maximal and explosive exercises (3 x 8 reps with 75% of 1 RM and 3 x 10 reps with 40% of 1 RM with 2min rest between the sets) and E was performed as continuous running with intensity between lactic and ventilatory threshold (60min).  $MVC_{max}$ , rapid force production as average force of 500ms ( $MVC_{500}$ ) and serum hormone concentrations (total testosterone and cortisol) were determined PRE, MID (following E or S, respectively) and POST loadings and repeated after recovery of 24h and 48h. Oxygen consumption was measured during the first and last 10 minutes of the endurance loading and running economy was determined as the average of minutes 6-8 and 56-58.

The main findings were significant decreases in  $MVC_{max}$  at MID and POST in men (MID, E+S, 8%,  $p<0.05$ ; S+E, 19%,  $p<0.001$ ; POST, E+S, 21%,  $p<0.001$ ; S+E, 19%,  $p<0.001$ ) in both loading conditions while these decreases were somewhat smaller in women (MID, S+E 14%,  $p<0.01$ ; POST, E+S, 12%,  $p<0.01$ ) Women did not show the same magnitude of reduction as men in  $MVC_{500}$  in both E+S and S+E). The recovery of  $MVC_{max}$  and  $MVC_{500}$  was faster in women, while in men reduced values were still observed at 48h of recovery following both loading conditions. Running economy was impaired in both men and women when endurance running was performed immediately after strength exercises (S+E). No significant changes occurred in serum testosterone in either men or women. During recovery serum testosterone at 24h and 48h of recovery was slightly decreased following S+E and slightly increased following E+S in men (at 24h, -14% vs. +7%, difference  $p<0.05$ ; at 48h, -8% vs. +16%, difference  $p<0.05$ ). Men showed slightly increased concentrations in serum cortisol ( $p=0.072$ ) at POST following S+E compared to E+S. This increase in serum cortisol in men was higher ( $p<0.05$ ) compared to unaltered serum cortisol concentrations in women.

In conclusion, the present results showed that the current loading protocol led to higher neuromuscular fatigue and larger serum cortisol responses in men than in women, which were in part accompanied by decreased concentration of anabolic hormones during the recovery phase in men when the strength loading was followed by the endurance loading. These findings might have important implications to optimize the combined strength and endurance loading regimes and its order as well as recovery from loading in recreationally endurance trained males and females.

**Key words: order effect, acute responses, combined endurance and strength loadings**

## **ACKNOWLEDGEMENTS**

This study was a cooperative effort between the University of Jyväskylä, Department of Biology of Physical Activity and the Research Institute for Olympic Sport (KIHU) and was carried out in the laboratories of the two institutes.

I would like to acknowledge the Department of Biology of Physical Activity and in particular my supervisor Keijo Häkkinen for providing the encouraging atmosphere towards my studies. Furthermore I would like to express my appreciation to Ritva Taipale for planning the realization of the study.

I also wish to thank the department's skilled technical staff (Pirkko Puttonen, Risto Puurtinen, Sirpa Roivas and Markku Ruuskanen) as well as the Research Institute for Olympic Sport (KIHU): Ari Nummela, Sirpa Vääntinen and Jussi Mikkola for their support during the measurements and results analysis. My appreciation also goes to a number of talented students who periodically participated in the study and the subjects, without their effort the study would have not been conducted.

# TABLE OF CONTENTS

ABSTRACT

ACKNOWLEDGEMENTS

TABLE OF CONTENT

1	INTRODUCTION .....	6
1	PHYSIOLOGICAL AND ENDOCRINE MECHANISMS RELATED TO RESISTANCE AND ENDURANCE EXERCISES .....	8
1.1	Oxygen and blood lactate kinetics in response to prolonged exercising.....	8
1.2	Hormonal mechanisms related to endurance and strength loadings .....	10
1.3	Endurance and strength training induced adaptations in blood lactate, running economy and serum hormone concentrations .....	12
1.3.1	Blood lactate and running economy.....	12
1.3.2	Serum hormone concentrations.....	13
2	ACUTE PHYSIOLOGICAL RESPONSES AND CHANGES IN FORCE PRODUCTION TO RESISTANCE AND ENDURANCE LOADINGS AND RECOVERY.....	16
2.1	Acute physiological responses and changes in force production to resistance loading and recovery .....	16
2.1.1	Blood lactate concentrations .....	16
2.1.2	Hormonal concentrations .....	17
2.1.3	Force production .....	21
2.2	Acute hormonal responses to endurance loading and recovery .....	21
2.3	Major gender differences in response to endurance vs. strength loadings .....	27
3	CHANGES IN PHYSIOLOGICAL VARIABLES AND FORCE PRODUCTION IN RESPONSE TO COMBINED ENDURANCE AND STRENGTH LOADINGS AND RECOVERY.....	30
3.1	Influence of endurance loadings on subsequent force production .....	30
3.2	Influence of resistance exercises on subsequent endurance loadings .....	32
3.3	Combined endurance and strength loadings and training.....	33
4	PURPOSE .....	36
5	RESEARCH HYPOTHESIS .....	38
6	METHODS.....	39
6.1	Subjects.....	39
6.2	Design.....	39
6.3	Strength and Endurance loading.....	41

6.4	Measurements.....	42
6.5	Training and nutrition.....	45
6.6	Statistical Analysis.....	45
7	RESULTS.....	47
7.1	Basal measurements.....	47
7.2	Endurance and strength performance.....	48
7.3	Maximal and rapid isometric force production.....	49
7.4	Blood lactate concentrations.....	50
7.5	Running economy.....	52
7.6	Serum hormone concentrations.....	53
7.6.1	Acute responses in anabolic serum hormone concentrations.....	54
7.6.2	Acute responses in catabolic serum hormone concentrations.....	56
7.7	Pearson-product-moment correlations.....	57
8	DISCUSSION.....	58
8.1	Acute changes and recovery in maximal and rapid force production.....	58
8.2	Acute changes in blood lactate concentrations.....	60
8.3	Acute changes in running economy.....	61
8.4	Acute changes and recovery of serum hormone concentrations.....	62
8.5	Limitations of the study.....	66
9	CONCLUSIONS.....	67
10	REFERENCES.....	68

# 1 INTRODUCTION

Strength and endurance training are often performed concurrently by elite and recreational athletes with the goal of improving performance capacity and work economy (Leveritt M & Abernethy 1999). Current exercise recommendations highlight the benefits of combined endurance and strength training for the development and maintenance of performance in both young highly trained (Paavolainen et al. 1999; Hoff et al. 2002; Mikkola et al. 2007) and recreational trained (e.g. Häkkinen et al. 2003) men and women. Nevertheless, adaptations to exercise loadings and the resultant performance improvements are specific to the type of activity performed (Hawley 2009). Thus, endurance and strength loadings performed separately lead to divergent acute responses and long-term adaptations (Kraemer et al. 1995).

Endurance training is aimed at increasing the rate of energy production from both aerobic and anaerobic pathways to improve the economy of motion, and increase maximum oxygen consumption ( $VO_{2max}$ , Hawley 2002). Thereby, accompanied acute hormonal responses include a change in concentrations of testosterone, growth hormones, insulin like growth factor I and cortisol. In addition, endurance exercises places some demands on force and power and may, therefore, acutely produce neuromuscular fatigue (Paavolainen et al. 1999).

Strength training, on the other hand, leads to increases in integrated electromyography which, in combination with hormonal adaptations, leads to significant increases in muscle mass, strength, and power (Kraemer & Ratamess 2004). The acute responses to resistance loading typically include acute decreases in strength, maximal neural activation and force-time characteristics of the muscles loaded (e.g. Häkkinen & Pakarinen 1995) with minor acute effects on the cardiorespiratory system (Kraemer & Ratamess 2004). In addition, endocrine responses to strength exercises typically include an acute increase in concentrations of anabolic as well as catabolic hormones.

Whereas reported findings in acute changes of neuromuscular and cardiovascular performance as well as endocrine variables have consistently been observed in men, acute changes in the endocrine system are typically more subtle and less consistent found in women (Shephard 2000; Fleck & Kraemer 2004).

Already a quarter-century ago, Hickson (1980) investigated the so called “interference effect” of

concurrent endurance and strength training performed over prolonged training periods, while recent studies have focused on the physiological and neuromuscular of both loadings performed on separate days (e.g. Kraemer et al. 1995; Bell et al. 1997; Häkkinen et al. 2003). The question of whether the order of endurance and strength training combined in a single session plays an important role with regard to acute responses of neuromuscular and endurance performance as well as on endocrine variables has received only limited scientific attention and its possible practical applications have yet to be determined.

# **1 PHYSIOLOGICAL AND ENDOCRINE MECHANISMS RELATED TO RESISTANCE AND ENDURANCE EXERCISES**

## **1.1 Oxygen and blood lactate kinetics in response to prolonged exercising**

The energy metabolism during physical activity is a complex system involving a large number of processes to supply energy for muscle contractions. Energy from macro-nutrient oxidation is transferred to the nucleotide molecule adenosine triphosphate (ATP) and stored in limited amounts in the muscle cells (Henriksson 2000). Cells must, thus, continuously resynthesize ATP at its rate of use (Åstrand et al. 1986) which can occur via three different pathways: formation of ATP by phosphocreatine breakdown, formation of ATP via the degradation of glucose or glycogen and the oxidative formation of ATP (Marieb 2004).

At sub-maximal intensities lactate production equals its oxidation and consequently the blood lactate level remains stable even though exercise intensity might slightly increase. Lactate production and accumulation, however, as exercise intensity increases and the muscle cells can neither meet the additional energy demands aerobically nor oxidise lactate as its rate of production (McArdle et al. 2007). As a result blood lactate concentration increases exponential in the exercising muscle which is commonly referred to as the blood lactate threshold (Åstrand et al. 1986) or more recently as the Onset of Blood Lactate Accumulation (OBLA, McArdle et al. 2007). The measurement of blood lactate concentrations appears, thus, to be an easy method for determining the intensity of physical exertion (Roecker et al. 2000).

Following strenuous exercise, blood lactate concentrations do not return to resting level immediately but may remain elevated above resting values for a certain period of time (Åstrand et al. 1985). Up to 60% of the accumulated lactic acid is aerobically metabolized while the remaining 40% is converted to glucose and protein and a small portion is excreted in the urine and sweat (Ingjer 1969). Active recovery has been shown to contribute to the elimination of excess blood lactate concentrations (Siegler et al. 2006).

In contrast to blood lactate accumulation, oxygen consumption ( $VO_2$ ) rises exponentially during the first minutes of exercise, followed by a plateau during the third and fourth minute and remains stable for the duration of effort (McArdle et al. 2007). Additionally, minute ventilation increases



linearly with  $\text{VO}_2$  and carbon dioxide production and, thus, reflects a balance between energy required by the working muscles and ATP production in aerobic metabolism.

However, induced by increased blood lactate levels with increasing exercise intensity, a change of blood pH occurs which causes carbon dioxide production to increase considerably and, thus, leads to a disproportional rise of minute ventilation in relation to  $\text{VO}_2$  (Figure 1). Pulmonary ventilation does then not link anymore to oxygen demand at the cellular level, a phenomenon which is called the ventilatory threshold (Roecker et al. 2000).

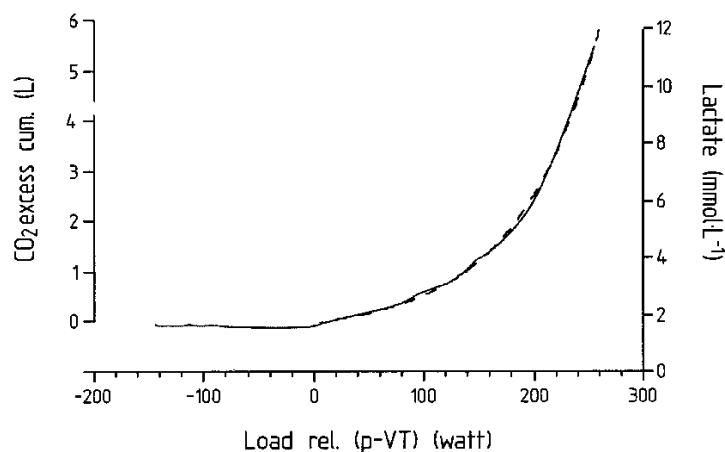


Fig. 1. Increase in blood lactate concentration and the accumulative excess carbon dioxide above the lactate ventilatory threshold, respectively (Roecker et al. 2000).

At maximal efforts oxygen consumption plateaus or increases only slightly with additional increases in exercise intensity. This represents the maximal oxygen consumption – also referred to as the maximal oxygen uptake or maximal aerobic power ( $\text{VO}_{2\text{max}}$ ). ATP cannot longer be resynthesized by oxidative formation or degradation of glucose or glycogen but via phosphocreatine breakdown which leads to a break off of exercise performance as anaerobic pathways can only be maintained for seconds, maximum minutes (McArdle et al. 2007).  $\text{VO}_{2\text{max}}$  therefore refers to the endurance capacity of an athlete and is among others used as a predictor of endurance performance.

*Running economy.* Even though  $\text{VO}_{2\text{max}}$  reflects endurance performance in heterogeneous groups of runners, it becomes less sensitive in homogeneous populations (Anderson 1996). Most of the time endurance performance is conducted at sub-maximal intensities and, thus, blood lactate levels remains constant. In addition, as at lower intensities only a fraction of  $\text{VO}_{2\text{max}}$  is consumed, running economy (RE) has been identified as an predictor of sub-maximal running performance and has

been traditionally described as the energy demand for a given velocity of sub-maximal running (Saunders et al. 2004). As shown in Figure 2, subjects with similar  $VO_{2max}$  values may actually consume different amounts of oxygen during steady state running. If body mass is taken into consideration, runners with good RE require less oxygen than runners with poor RE for the same velocity and distance (Palmer & Sleivert 2001).

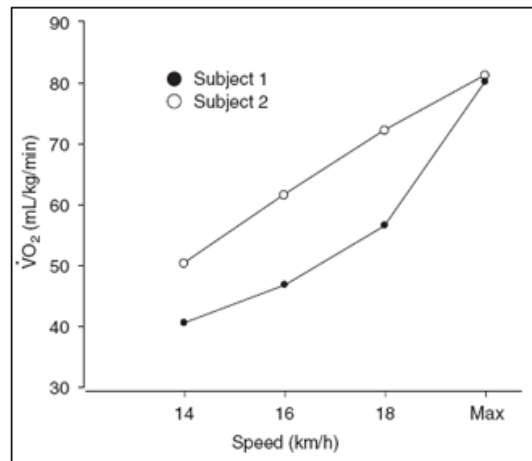


Fig. 2. Running Economy in subjects with equal  $VO_{2max}$ . Subject one requires less oxygen for the given velocity than subject two and can therefore be considered as being more economical (Saunders et al. 2004)

*Excess post exercise oxygen consumption (EPOC)*. As with blood lactate kinetics, oxygen consumption does not return to resting level immediately following various modes of exercise but may remain elevated above resting values for a certain period of time (Drummond et al. 2005). According to Gaesser & Brooks (1984) this has been described as excess post exercise oxygen consumption (EPOC) and has classically been referred to as the oxygen debt or recovery oxygen consumption. Recent publications have shown that both resistance (Nagasawa 2008) and endurance training sessions (Borsheim et al. 2003) have a considerable impact on the post-exercise oxygen consumption and might, thus, effect subsequent training sessions when no or too short recovery is provided.

## 1.2 Hormonal mechanisms related to endurance and strength loadings

Hormones are defined as chemical mediators that regulate the metabolic function of other cells in the body (Marieb 2004). More detailed, hormones are produced by specific host glands belonging to the endocrine system from where they are released into the blood to be transported throughout the

body and bind to specific target cells. In addition to the nervous system the endocrine system, thus, plays a major role in coordinating and integrating the activity of body cells. H

Hormones can be generally grouped into anabolic and catabolic steroids as well as amino-acid based hormones and eicosanoids (Griffin and Ojeda 2004). Within the context of sport and exercise science this becomes important as anabolism generally refers to the built up of structures (e.g. bonding together of amino acids to generate proteins) and, therefore, plays a major role in muscle growth (Bhasin et al. 2001). Catabolic hormones, in contrast, are characterized by opposite effects resulting in a breakdown of complex cell structures and are, thus, associated with physiological stress (Fleck & Kramer 2004).

In general, hormonal reactions lead to widespread diverse effects including integration and regulation of bodily functions and, therefore, provide stability to the body internal environment. More specific the major functions of hormones can be summarized as the activation of enzyme actions, cause of muscular contractions and relaxations, stimulation of protein and fat synthesis and augmentation of body responses to physical and psychological stress (McArdle et al. 2007). Circulating levels of hormones, however, do not necessarily reflect changes in physiological function of cellular and sub-cellular levels. The physiological impact of altered hormonal concentrations is not observed until it has initialized cellular responses. These cellular responses are, thus, dependent on availability and sensitivity of hormone receptors as well as the availability of substrates and materials for adaptive responses (Tremblay & Chu 2000). Specific responses of target cells may, therefore, be observed after a period of seconds, minutes or even days (Keizer 1998).

Physical exercise is known as a powerful stimulus for the endocrine system (Karkoulias et al. 2008). According to Keizer (1998), physical activity of moderate to high intensity (e.g. endurance loadings) is able to elicit remarkable changes in stress hormone secretion which is important in order to augment muscle enzyme activities and, thus, energy release and expenditure. Caution must, however, be paid since changes in hormonal concentrations following physical activity do not necessarily reflect alterations in hormonal secretion and elevation but can be due to fluid volume shifts, tissue clearance rates, hormonal degradations, venous pooling of blood, interactions with binding proteins in the blood as well as due to receptor interactions (Fleck & Kraemer 2004).

Within the context of endurance and strength loadings several hormones have previously been

studied (Consitt et al. 2002). These include changes in anabolic hormonal concentrations such as testosterone (Tremblay et al. 2005; Daly et al. 2005), growth hormones (Näveri et al. 1985; Kokalas et al. 2004) and circulating insulin like growth factor 1 (Nguyen et al. 1998; Chicharro et al. 2001) as well as catabolic hormones such as cortisol (Virus et al. 1996; Karkoulas et al. 2008).

### **1.3 Endurance and strength training induced adaptations in blood lactate, running economy and serum hormone concentrations**

Both endurance and strength training have been shown to induce wide spread adaptations in the musculoskeletal system, the pulmonary system and the endocrine system (Hawley, 2002). These adaptations include enhanced oxygen transportation capacities, improvements in working economy and reductions of blood lactate concentrations at sub-maximal intensities (McNicol et al. 2009). Consequently lower oxygen demands during exercise leads to a faster decline in oxygen consumption post-exercise and, thus, enhance the ability of recovery (Gaesser & Brooks 1984). Chronic adaptations can, thus, occur in both basal concentrations as well as in acute responses to exercise stimulus and recovery.

It has been suggested that prolonged high-volume and low intensity training periods may cause long term endocrine adaptations (Grandys et al. 2009). Previously untrained subjects, thus, elicit more profound endocrine changes than their trained counterparts (Gulledge et al. 1996). However, in both untrained and trained subjects the adaptations seems to be divergent between both endurance and strength training as endurance training has been shown to elicit more profound catabolic changes compared to resistance training (Consitt et al. 2002).

#### **1.3.1 Blood lactate and running economy**

According to McCrae et al. (1992) prolonged endurance training performed over several weeks at intensities below the lactate threshold leads to a decrease in blood lactate concentrations and, thus, a delayed onset of blood lactate accumulation (OBLA). As a consequence, blood pH can be maintained for a prolonged period resulting in increased endurance performance.

Spengler et al. (1999) found decreased blood lactate concentrations after 4 weeks of specific respiratory endurance training (Figure 3). The mechanisms underlying these effects may be related to both a decrease of the rate of lactate formation and simultaneously increase of rate of lactate removal during the loading (McArdle et al. 2007). Accompanied physiological adaptations include a

greater number of increased cell lactate transporters which enhances the exchange and removal of lactate from the blood (Ashleigh et al. 2009) and an increase in mitochondria enhancing skeletal muscle oxidative capacities (Spengler et al. 1999).

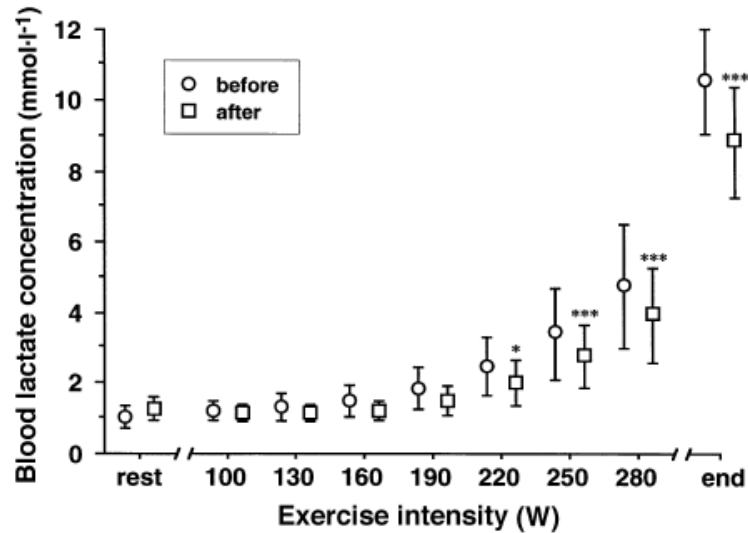


Fig. 3. Blood lactate concentrations at rest and during an incremental endurance test after 4 weeks of respiratory endurance training. \* $p < 0.05$ , \*\*\* $p < 0.01$  (Spengler et al., 1999).

Whereas the mechanisms underlying the shift of OBLA are similar during endurance and strength training, the latter one has been shown to be less effective in reducing blood lactate accumulation. Nevertheless, Warren et al. (1992) showed positive adaptations to concentrations of blood lactate after already one week of high volume resistance training. These adaptations seem to, however, be clearly dependent on the mode of training performed (Kraemer & Ratamess 2004).

### 1.3.2 Serum hormone concentrations

Vuorimaa (2007) investigated anabolic and catabolic hormone responses to continuous and intermittent running protocols in middle- and long distance runners and found significant higher responses to intermittent running loadings in middle distance runners. This finding was suggested to be the result of different training strategies, as marathon runners prefer slower continuous type of running. Similarly, Fleck & Kraemer (2004) reported acute serum hormone concentrations in response to resistance loadings to be dependent on the training background of the subjects.

*Testosterone.* Serum testosterone concentrations have been typically used as a physiological marker

to evaluate the anabolic status of the body. Testosterone promotes muscle hypertrophy by enhancing protein synthesis and may also contribute to force production by its potent influence on neural mechanisms (Fleck & Kraemer 2004).

Hackney et al. (2003) reported that basal testicular testosterone production in endurance-trained men is lower compared to untrained subjects. This might be caused by the fact that endurance athletes may need less testosterone and, therefore, maintain a reduced basal level of overall muscular mass development to improve endurance performance. Grandys et al. (2009), in contrast, found increased basal concentrations of total testosterone by 17% and free testosterone by 26% after 4.5<sup>-1</sup> wk of cycle endurance training at 90% of power output from the predicted lactate threshold. Taking both findings together one might suggest a biphasic adaptation of serum testosterone production with increased basal concentrations after a short period of training, followed by reduced basal levels after month and years.

A similar trend was shown for strength trained men performing 21 weeks of hypertrophy training (Ahtiainen et al. 2003). Free testosterone concentrations were increased after 14 weeks of training but were not significantly different compared to baseline after 21 weeks. Similar results, though smaller total values, were found for previously untrained subjects indicating the role of training-status with regard to endocrine adaptations. Testosterone concentrations in strength compared to endurance athletes are, however, generally expected to be higher (Kraemer et al. 1995).

*Growth hormones and IGF I.* Growth hormone (in particular the main circulating isoform 22 kd) and IGF 1 have anabolic effects on the muscle cell by increasing transportation of amino acids into the muscle cells and increasing protein synthesis (Griffin & Ojeda 2004).

The basal concentrations of growth hormones and IGF-1 have been reported to be slightly increased or maintained after several weeks of intense strength and/ or endurance training (Weltman et al. 1992). Manetta et al. (2003) showed that 4 months of intensified cycle training in professional cyclists was not sufficient to elicit changes in IGF-1 concentrations while concentrations of IGF binding proteins 1 and 3 were increased. Similar findings have been also shown in response to strength training. Kraemer et al. (1999) suggested this to reflect a low adaptability of IGF-1 to chronic strength training. Although the reasons are not clearly investigated yet, the IGF binding protein 3 (IGFBP-3) seems to be more sensitive to intensive strength and/ or endurance training.

*Cortisol.* Cortisol, originally named as a glucocorticoid, influences the metabolism of amino acids and glucose. In the fasted state cortisol helps to maintain blood glucose levels by stimulating gluconeogenesis and peripheral release of substrates (Fleck & Kraemer 2004).

Cortisol concentrations generally reflect the long term training stress. It has been suggested that cortisol levels are higher in endurance trained athletes compared to sedentary controls (Tegelman et al. 1990) and strength trained subjects (Kraemer et al. 1995). Nevertheless, studies of Filaire et al. (1996) and Purge et al. (2006) showed a considerable decrease after 24 weeks of rowing training in men. Similarly, results regarding cortisol concentrations following prolonged periods of resistance training are also not consistent. No change (Fry et al. 1994) as well as decreases (Häkkinen et al. 1985; Kraemer et al. 1998) and increases (Häkkinen & Pakarinen 1991) have been observed in previously untrained versus already well trained strength athletes.

## 2 ACUTE PHYSIOLOGICAL RESPONSES AND CHANGES IN FORCE PRODUCTION TO RESISTANCE AND ENDURANCE LOADINGS AND RECOVERY

### 2.1 Acute physiological responses and changes in force production to resistance loading and recovery

Resistance exercises of various modes have been shown to acutely induce changes in hormonal concentrations (Häkkinen & Pakarinen 1995) and blood lactate accumulation (Kang et al. 2005) in both men and women. These immediately responses are highly dependent on the type of resistance exercise performed, i.e. number of sets and repetitions per set, length of the rest period and muscle mass involved (Linnamo et al. 2005; Ahtiainen et al. 2003a). According to Kraemer & Ratamess (2004), it needs to be distinguished between hypertrophy type of resistance loadings (6-12 repetitions, 70-80% 1RM, resting period 60 – 90 seconds), maximum strength loadings (1-6 repetitions, 80-85% 1RM, resting period 120-180 seconds) and explosive type of strength loadings (8-10 repetitions, 30-60% 1RM, resting periods around 180 seconds). In addition, strength-endurance loadings are commonly performed and include a greater number of repetitions of lower intensities, combined with a short resting period (60-70% 1RM) (Smilios et al. 2007).

#### 2.1.1 Blood lactate concentrations

Blood lactate responses to 3 different types of resistance loading protocols are presented in figure 4.

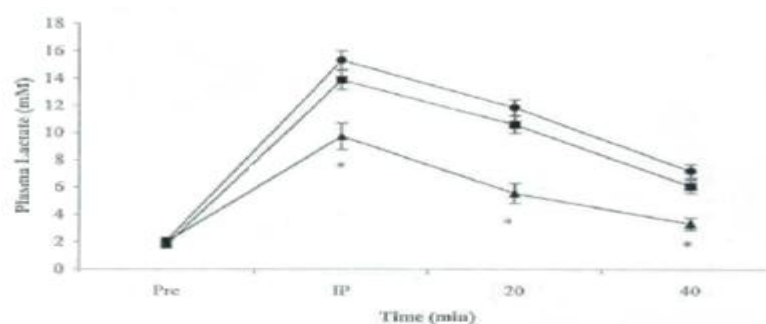


Fig. 4. Acute plasma lactate concentrations to 3 different resistance exercise protocols. ● 60% 1RM, 15 repetitions, ■ 75% 1RM, 10 repetitions, ▲ 90% 1 RM, 4 repetitions. Pre refers to pre-exercise measurements, IP mean immediately post exercise, and 20 and 40 refers to minutes of recovery (Kang et al. 2005)



It has been shown that strength-endurance exercises with a high volume and short resting periods lead to higher blood lactate accumulations compared to hypertrophy and maximum strength exercise protocols (Kang et al. 2005). Furthermore, loadings that are rather aiming for muscle hypertrophy seems to acutely produce higher blood lactate concentrations compared to loadings of lower, sub-maximal intensities (Raastad et al. 2000).

Explosive strength loadings may not show acute blood lactate responses as high as observed during heavy resistance exercises (Linnamo et al. 2005; McCaulley et al. 2009) which reflects the importance of the exercise mode. Resistance protocols of heavy loads combined with a large number of repetitions and a low resting period between the sets may disturb homeostasis to a greater extent than aerobic exercises and, thus, require a longer recovery period, shown in higher lactate accumulations (Tesch et al. 1986; Kang et al. 2005).

Following an acute bout of resistance exercise accumulated blood lactate concentrations are quickly removed. The half-life of the lactic acid portion of the oxygen debt has been suggested to be approximately 25 minutes (Hermansen et al. 1976). Thus, approximately 95% of the accumulated blood lactate is removed from the blood within 1 hour and 15 minutes.

### **2.1.2 Hormonal concentrations**

There is strong evidence that blood lactate may stimulate testosterone and growth hormone responses, indicating highest changes in concentrations of these hormones in response to metabolic stress induced by heavy resistance loadings (Raastad et al. 2000). Table 1 gives an overview over acute changes in concentrations of serum testosterone, growth hormones and serum cortisol in response to various resistance loading protocols.

*Testosterone concentrations.* Highest concentrations of serum testosterone are typically observed following heavy resistance (hypertrophy) loadings characterized by high metabolic stress. The interaction of a resistance loading session's intensity and volume affects the acute testosterone responses. The magnitude of acute serum testosterone responses can, thus, be seen to reflect the magnitude of the stress of the exercise session (Häkkinen & Pakarinen, 1995). As with blood lactate concentrations, neural type of resistance loadings protocols such as explosive exercises and maximal strength loadings characterized by prolonged resting periods and lower loading are not sufficient enough to induce remarkably changes in serum testosterone concentrations (Linnamo et al. 1998).

Table 1: Acute serum testosterone (TT), growth hormone (GH) and serum cortisol (C) responses to various resistance protocols in young men. ↑ indicates increase, - indicates no change and ↓ refers to decreased concentrations, N/A means values not given.

Reference	Exercise type	Intensity	Sets	Repetitions	Rest (min)	Hormonal responses		
						TT	GH	C
Kraemer et al. 1991	Total body workout	5RM 10RM	8	5 10	3 1	↑	↑	N/A
Häkkinen & Pakarinen (1995)	Bench press, sit-ups, bilateral leg press	100% 10RM	5	5	3	↑	↑	↑
Raastad et al. (2000)	Squat Front squat Knee extension	100% 3RM 100% 3RM 100% 6RM	3	3 3 6	4	↑	↑	-
	Squat Front squat Knee extension	70% 3RM 70% 3RM 76% 6RM	3	5 4 2	4	-	↑	↓
Ahtiainen et al. (2003)	Leg press Squat Knee extension	100% 12RM	4 2 2	12	2	↑	↑	↑
	Leg press Squat Knee extension	115% 12RM (assisted)	4 2 2	12	2	↑	↑	↑
Linnamo et al. (2005)	Sit ups bench press bilateral leg extension	100% 10RM	5	10	2	↑	↑	N/A
		70% 10RM	5	10	2	-	↑	N/A
		40% 10RM	5	10	2	-	↑	N/A
Smilios et al. (2007)	Seated chest press, pec deck, lateral pulldowns, biceps curls, leg extension and leg flexion	60% 1RM	3	15	1:30	↑	↑	↑
McCaulley et al. (2009)	Back squat	75% 1RM	4	10	1:30	↑	N/A	↑
	Back squat	90% 1RM	11	3	5	↑	N/A	↓
	Jump squat	Max power	8	6	3	↑	N/A	↓

The exercise induced increase in serum testosterone concentrations has been shown to be followed by slight reductions in testosterone levels during a post-loading recovery period with lower GH values reported after 15 and 30 min (Ahtiainen et al. 2003a) as well as after 2 hours (Häkkinen & Pakarinen 1995; Raastad 2000). In a study of Häkkinen & Pakarinen (1994) serum testosterone

concentrations were declined for up to 48h, indicating the extreme exercise induced stress and, thus, a prolonged recovery time from an endocrinological perspective.

*Growth hormone concentrations.* Most studies provided evidence that growth hormone (GH) reacts in a similar manner as testosterone in response to resistance loadings. According to Häkkinen & Pakarinen, (1995) and Linnamo et al. (2005) the overall load as well as the work volume and frequency of the exercises seem to determine the magnitude of the GH response (Figure 5).

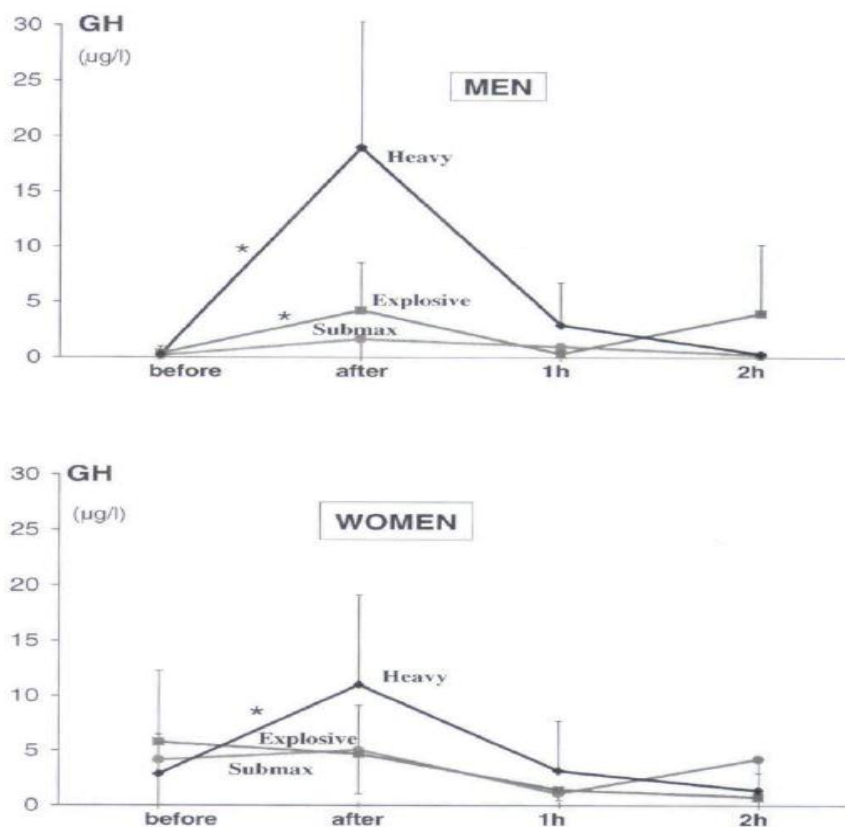


Fig. 5 Acute responses of GH concentrations in men and women following submaximal, explosive and heavy type of strength loadings. Values are given in mean $\pm$ SD, \* refers to significant differences ( $p < 0.05$ ) (Linnamo et al. 2005).

Only Raastad et al. (2000) did not find acute increases in GH following a loading protocol with lower intensities. The authors of this study explained their findings with longer resting periods, which might induce fatigue at a lower rate. It might be, thus, suggested that during lower intensities the role of resting periods becomes more important in order to show acute increases in serum GH concentrations. In addition, although the lactate differences observed for resistance loadings of 100% and 70% in the study of Raastad et al. (2000) were great, no differences in GH concentrations

were observed, indicating that in contrast to serum testosterone levels, blood lactate accumulation and resulting changes in blood pH are not necessarily affecting acute responses of GH.

Following a strenuous bout of resistance exercise, serum growth hormone concentrations have been shown to decrease immediately following the end of the loading. While after 15min and 30min still significant increased concentrations may be observed (Ahtiainen et al. 2003a), after 2 hours GH concentrations close to baseline levels have been reported (Häkkinen & Pakarinen 1995; Raastad et al. 2000)

*Insulin like growth factor 1 concentrations:* Results regarding the responses of IGF-1 and its binding proteins are limited. Although not completely understood, it has been suggested that some of the effects of growth hormone are mediated by stimulating the cell released insulin like growth factors (Florini et al. 1996). It is, thus, expected that in particular IGF-1 reacts in a similar pattern as GH in acute response to resistance loading sessions. Early results of Kraemer et al. (1992) suggested serum IGF-1 concentrations to be acutely increased following a whole body workout of 10 repetitions with 1min rest between the sets whereas similar concentrations following 5RM and 3min rest were observed only after 60minutes recovery. Further investigations of Kraemer et al. (1995) showed that levels of IGF-1 seem to be dependent on physiological factors such as metabolic clearance rates and the release of IGF-1 from other non-hepatic cells (e.g. fat or muscle cells) caused by tissue disruption from exercise. The impact of resistance loadings may, thus, not be in the alteration of IGF-1 levels but rather in alterations to individual components of the IGF-1 system, e.g its binding proteins (Nindl et al. 2001). Serum IGF-1 concentrations are, therefore, dependent on the exercise intensity and might peak not immediately after the resistance loading but in the later phase of recovery.

*Cortisol concentrations:* Highest concentrations in serum cortisol have been shown in acute response to resistance loadings using heavy weights which induce high metabolic stress (e.g. Ahtiainen et al. 2003a). Following maximal strength protocols, on the other hand, cortisol levels remained the same (Raastad et al. 2000) and were decreased after an explosive bout of resistance exercise (McCaulley et al 2009). As the authors did not explain their findings, the cause of reduced cortisol concentrations in response to explosive loadings remains unclear. It might be, however, concluded that as with other stress hormones, cortisol concentrations are related to the exercise mode performed and in particular sessions of high total work with short rest intervals between the sets seems to yield the highest accumulation (Smilios et al. 2007).

Data regarding serum cortisol kinetics during prolonged recovery are, unfortunately, lacking. It has been, however, reported that in response to a heavy resistance loading protocol, serum cortisol concentrations appeared to return to baseline levels or even below within 2 hours (Häkkinen & Pakarinen 1995).

### **2.1.3 Force production**

Heavy resistance strength loadings may lead to acute neuromuscular fatigue reflected in both reductions in the maximal voluntary neural activation as well as in maximal force of the exercising muscles. Decreases in maximal force of 25% have previously been observed in men following a strenuous neural type of resistance loading protocol of 20x1x100% 1 RM loads (Häkkinen 1993). The magnitude of neuromuscular fatigue, however, seems to be depending on the volume and intensity of the strength loading protocol as well as on the exercise mode performed (Häkkinen 1994; Linnamo 1998). A hypertrophic type of loading with maximal weights during 4x12RM and 2 min rest between the sets led to drastically greater decreases in maximal isometric force of up to 40% (Ahtiainen et al. 2003a) while by increasing the load in the same study further so that assistance was necessary to complete the set, acute reductions greater than 50% have been observed. The values in studies by both Häkkinen (1993) and Ahtiainen et al. (2003a) remained reduced for 2 days in the normal protocol and was still reduced post 72h following the assisted type of loading.

Similarly to reductions in maximal force production, significant decreases in explosive power (Linnamo et al. 1998) as well as in the rapid portion of force production during maximal isometric force (Häkkinen 1993) have been observed.

## **2.2 Acute hormonal responses to endurance loading and recovery**

Similarly to resistance loading induced acute changes in hormonal concentrations, the endocrine system sensitively responds to endurance loadings depending on their duration and intensity (Tremblay et al. 2005). Exercise intensity has been suggested to influence acute changes in anabolic hormone concentrations such as serum testosterone, serum growth hormone, serum IGF-1 and serum cortisol concentrations (Stokes et al. 2002a; Kokalas et al. 2004; Tremblay et al 2005).

*Testosterone concentrations.* Tremblay et al. (2005) systematically investigated the effect of incremental endurance exercises on steroid concentrations in male subjects and found a significant

relationship between acute changes in testosterone concentrations and exercise duration. Both free and total testosterone levels showed an initial increase of about 20% in the first hour of an 80min and 120min run of low intensity with a subsequent decline which continued during the recovery. In addition, 40min of running at the same intensity affected serum testosterone concentrations to a lower extent, indicating a strong association between testicular response and exercise duration.

Testosterone concentrations seems, thus, to considerable increase after 40min of exercise (Tremblay et al. 2005), might show peak values between 60 and 120min (Daly et al. 2005) and show a significant decline after marathon or ultra marathon distances, longer than 4 hours (Figure 6, Kuoppasalmi et al. 1981; Kraemer et al. 2008; Karkoulas et al. 2008).

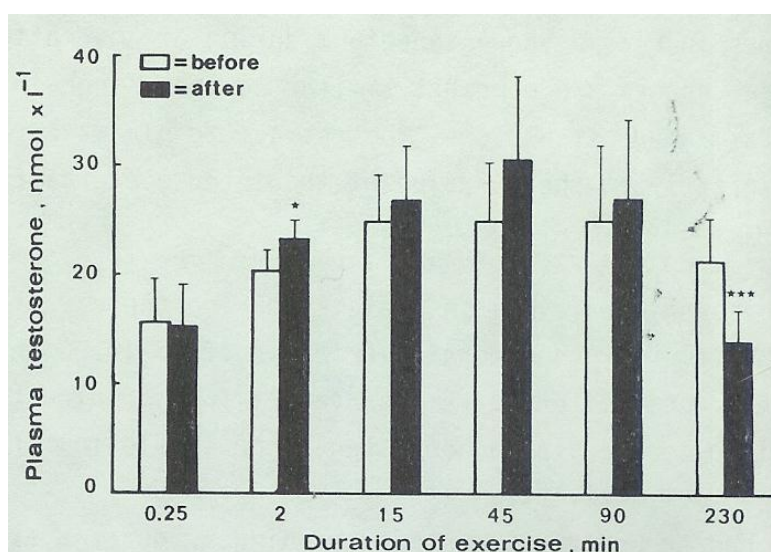


Fig. 5. Plasma testosterone concentrations before and after sprint and endurance running loadings of different intensities and durations. \* indicates significant difference ( $p < 0.05$ ), \*\*\* indicates significant difference ( $p < 0.01$ ) (Kuoppasalmi 1981)

According to Kokalas et al. (2004), this suggests that endurance loadings performed over shorter durations may promote anabolic processes and, although this does not necessarily mean muscle hypertrophy, it might mediate increased expression of aerobic enzymes or adaptations in other processes. Speculative reasons for decreased post-exercise levels of testosterone in response to prolonged endurance exercises were given by Kraemer et al. (2008), who assumed that either the rate of testosterone utilisation increased to exceed production during the race to preserve protein tissue or the rate of production decreased during the race because of inhibitory mechanisms.

Table 2: GH responses to endurance loadings of various durations and intensities in moderate trained athletes. Subscribed numbers refers to different groups within one study.

Study	Number of subjects, gender and training-status of subjects	Measured specimen	Endurance loading	Loading induced GH response (compared to baseline)	GH concentration during recovery (compared to baseline)
			Intensity		
Näveri et al. 1985 <sup>1</sup>	N=5, males, moderate trained	Venous blood sample	Outdoor running	No significant increase	Not significant different after 3 hours
			4.2-4.3min·km <sup>-1</sup>		
			90min		
Näveri et al. 1985 <sup>2</sup>	N=5, males, moderate trained	Venous blood sample	Outdoor running	6 fold increase	Not significant different after 3 hours
			3.2-3.3min·km <sup>-1</sup>		
			45min		
Pritzlaff et al. 1991	N=10, males, moderate trained	Venous blood sample	Treadmill running	Positive relationship to exercise intensity	Not significant different after 90min at all intensities
			5 different intensities		
			30min		
Kokalas et al. 2004 <sup>1</sup>	N=6, males, elite athletes	Venous blood sample	Rowing ergometer	504% increase	Not significant different after 4 hours
			3-4mmol·l <sup>-1</sup> lactate		
			60min		
Kokalas et al. 2004 <sup>2</sup>	N=6, males, elite athletes	Venous blood sample	Rowing ergometer	309% increase	Not significant different after 4 hours
			4x5min, 5min rest		
			5-6mmol·l <sup>-1</sup> lactate		
Kraemer et al. 2008	N=16, males, moderate trained	Venous blood sample	Running: N=10 Cycling: N=6	~30 fold increase	N/A
			N/A		
			Runners: mean±SD: 33.98±6.12h Cyclists: mean±SD: 21.83±6.27h		

Less consistent seem to be, however, the testosterone kinetics during recovery which might partly be related to the conducted study designs. Whereas some studies obtained follow up measurements within several hours, other investigations reported recovery values only after days or even weeks (Karkoulas et al. 2008). Kuoppasalmi et al. (1980) found significant decreased testosterone levels up to 6 hours post-exercise, following 45-90min of distance running while after 24 hours plasma testosterone levels were almost similar to baseline values. Although the reasons for that are yet not

clearly investigated, current investigations confirmed these findings.

*Growth hormone concentrations.* Most of the available information regarding GH kinetics in acute response to exercise loadings are available from studies investigation short-term sprint loadings. Table 2 presents conducted studies dealing with the relationship of endurance loadings and GH concentrations.

In addition to Pritzlaff et al. (1999), Table 2 clearly shows a threshold relationship between intensity of exercise and the measured GH concentrations in response to endurance loadings. The GH response to exercise seems to be relatively attenuated until an exercise intensity equal or greater than the lactate threshold, indicating a linear dose-response relationship between exercise intensity and the GH levels, with highest levels of GH observed immediately post-exercise (Figure 7).

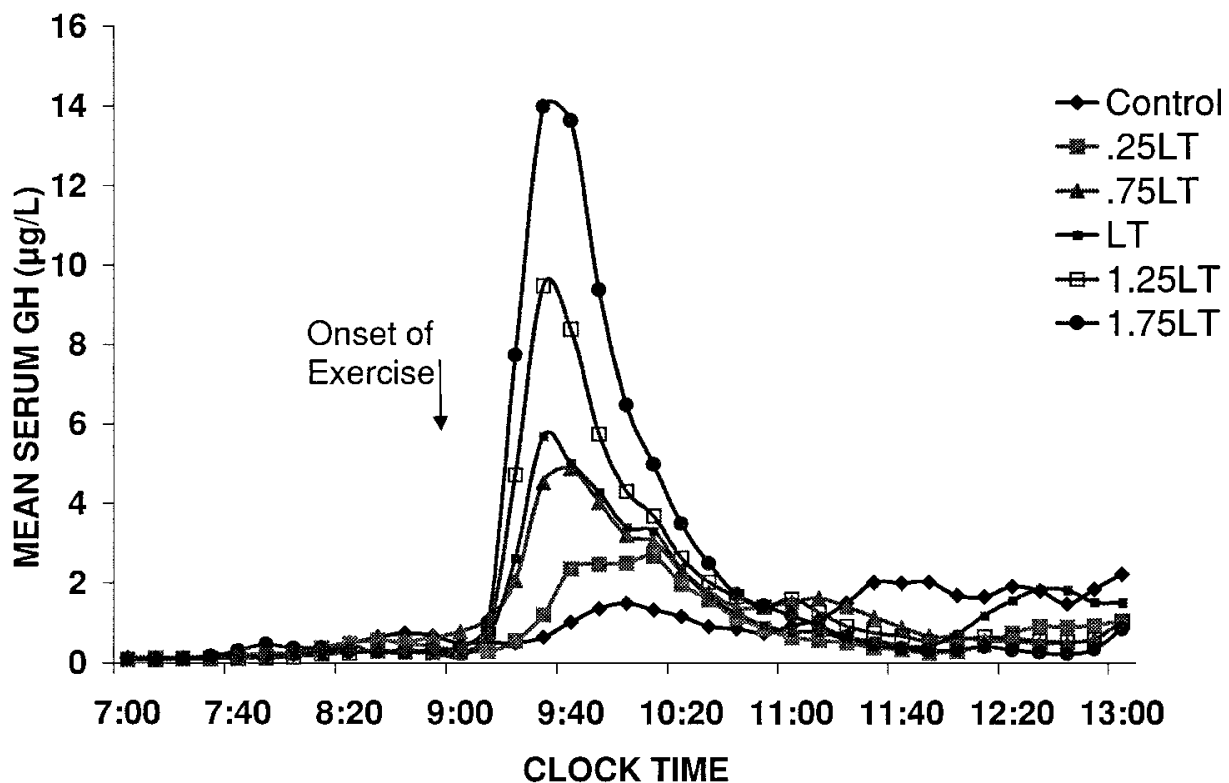


Fig. 6. GH responses to 30 minutes treadmill running at different constant intensities. .25LT and .75LT refers to 25% and 75% of the difference between O<sub>2</sub> consumption at lactate threshold and O<sub>2</sub> consumption at rest. 1.25LT and 1.75LT refers to 25% and 75% of the difference between at O<sub>2</sub> lactate threshold and peak O<sub>2</sub> (Pritzlaff et al. 1999).



Regarding the GH concentrations during recovery, the available data provide evidence that resting levels are, similarly to GH concentrations in response to heavy resistance loadings, reached within 1 – 3 hours following loadings of different intensities. However, unfortunately follow up measures in the presented studies were inconsistent and thus accurate recovery kinetics cannot be concluded.

*Insulin like growth factor 1 concentrations.* Although IGF 1 is regulated by the GH system, the acute IGF 1 responses to endurance loadings are not attenuated by increasing the alkalosis of the blood making it difficult to conclude changes in the IGF-system from elevated GH concentrations (Kraemer et al.2000). In contrast to endurance loading induced changes of T and GH concentrations, data on changes on IGF-1 is lacking.

Figure 8 indicates an increase of total IGF-1 by 12% at the end of an incremental cycling protocol to voluntary exhaustion. In contrast, 60km of ski racing seems to acutely reduce totally IGF-1 concentrations by 15% and again contrary are the responses of IGF-1 levels to a 90min simulated soccer game which did not show changes compared to baseline values (Nguyen et al. 1998).

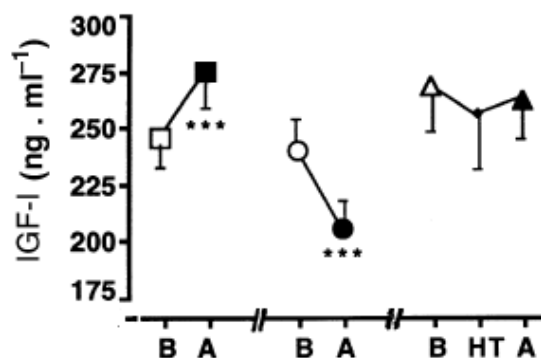


Fig. 7. Plasma IGF 1 responses before (A) and after (B) three different types of exercise. Left hand site: incremental cycle test; Centre: 60Km cross country ski competition; Right hand site: 90min simulated soccer game. HT indicates half-time measurements, \*\*\* significant different from baseline values (modified from Nguyen et al. 1998).

Collectively these results suggest an intensity related correlation of IGF-1 to continuous endurance loadings with a speculative biphasic response (Nguyen et al. 1998). This might be related to blood lactate kinetics during exercise, as IGF-1 has been shown to be sensitive to lactic acid concentrations (Kraemer et al. 2000).

*Cortisol concentrations.* Endurance exercise of longer durations such as marathon and ultra-

marathon distances may create a rather catabolic metabolism and, thus, an increase in cortisol concentrations during and immediately after the loading (Karkoulias et al. 2008; Kraemer et al. 2008). There seems to be a consistent relationship between intensity and duration of the endurance loadings and acute changes in cortisol concentrations. Viru et al. (1996) found increased cortisol concentrations in response to endurance loadings only above an intensity threshold of 60-70% of  $VO_{2max}$ . Tremblay et al (2005), in contrast, observed a considerable increase in cortisol concentrations already at lower intensities after a duration exceeding 80min supporting the theory that elevations of cortisol concentrations are dependent on both exercise intensity and duration. According to Karkoulias et al. (2008), this might be due to a stimulation threshold of the pituitary-adrenal axis which controls the reaction of cortisol secretion in response to stress.

It is, however, noticeable that endurance loadings, lasting less than 1 hour seem to be not sufficient to stimulate excessive cortisol secretion (Kokalas et al, 2004). As in a study of Tremblay et al. (2005) low intensity and high volume endurance exercise led to significant increases of cortisol concentrations, while higher intensity exercise over a shorter duration did not show significant changes (Kokalas et al. 2004), it can be suggested that the loading duration plays a major role. This was confirmed by Vuorimaa (2007) who reported that in continuous (aerobic) type of running the increase in cortisol takes place earlier if the intensity is higher.

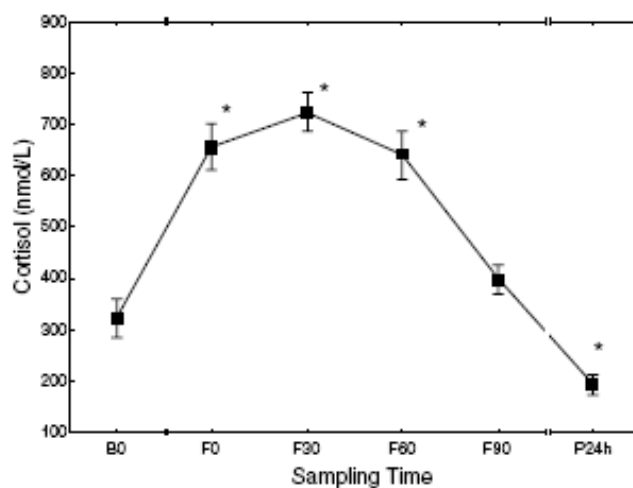


Fig. 8. Acute changes and recovery of cortisol concentrations in relation to strenuous endurance exercise. Recovery measures were taken after 30, 60 and 90min as well as after 24hours. \* Significant different from baseline (Daly et al. 2005)

Collecting these results together, a general trend of cortisol concentrations in response to endurance loadings can be concluded and is shown in Figure 9. There appears to be a biphasic increase in circulating concentrations of cortisol during endurance loadings. It has been, thus, suggested that the first increase occurs during the first 10-20min of exercise which followed by a second peak within few minutes post-loading (Daly et al. 2005). Following the highest peak of cortisol concentrations, values were continuously decreased up to significant lower concentrations compared to baseline values, measured 24hours post loading.

### **2.3 Major gender differences in response to endurance vs. strength loadings**

*Blood lactate accumulation.* Lactate thresholds are altered by prolonged endurance and strength training and previous research has suggested that the mechanisms are similar in both men and women (Henriksson 2000). The results regarding changes in lactate concentrations in acute response to submaximal and maximal bouts of exercises are, however, controversial.

Several studies found blood lactate concentrations in response to endurance and strength loadings to be different between men and women with higher absolute values observed in men (Sanchez et al. 1980; Brooks et al. 1990; Gratas-Delamarche et al. 1994). As the lactate clearance rate was not analysed in these studies, faster lactate elimination in women cannot be excluded. In the study of Gratas-Delamarche et al. (1994), lower blood lactate concentrations in responses in women in response to a Wingate test were associated with lower workloads and energy output. The ratio between blood lactate and Watts expressed per kilogram of lean body mass, however, was similar in men and women.

*Force production.* Häkkinen (1993) observed decreases in maximal force production in response to a heavy set of resistance loading (20x1x100%) to be greater in men than in women. The early recovery of maximal force after the same protocol was somewhat faster in women compared to men but maximal force was still reduced in both genders following two days of recovery. Women, thus, usually develop less fatigue than men (Kraemer & Häkkinen 2002).

Similar differences between sexes were also observed following a hypertrophic type of resistance loadings in which men showed still reduced values after 24h of recovery while women were almost recovered (Häkkinen 1994). In addition, women have been shown to elicit less fatigue in response to explosive resistance loadings as well as smaller reductions in the rapid portion of force production as shown in the average force-time curve over 500ms (Häkkinen 1993; Kraemer &

Häkkinen 2002).

*Hormonal concentrations.* Increased hormonal concentrations in acute response to both strength and endurance loadings are dependent on the individual basal hormonal concentrations and again these are dependent on gender (Consitt et al. 2002; Weltman et al. 2006; Gilbert et al. 2008). Subject samples that are not matched for gender, thus, demonstrate an increased outcome variance (Hackney et al. 2008). Research designs aimed to show hormonal responses to all kind of exercise loadings should include populations matched for sex.

Until puberty, basal hormonal concentrations are somewhat similar between boys and girls (Tremblay & Chu 2000). Thereafter, testosterone production at rest is much greater in men (Hackney et al. 2008). In contrast, women maintain higher GH concentrations than men at all ages and manifest less orderly patterns of pulsatile GH release (Nevill et al. 1996; Wideman et al. 1999). Moreover, the higher GH secretion rate in women might reflect a greater mean mass of GH secreted per burst compared with men during rest.

Similar findings were also reported by Weltman et al. (2006) who found higher GH secretion rates at different exercise intensities in young and older women compared to male subjects but similar comparable pulsatile hormone release, indicating higher baseline values in women. It can be suggested that this is related to the combined anabolic effects exerted by testosterone and GH on target tissues as serum and free testosterone concentrations in the same study were significantly greater in men than in women.

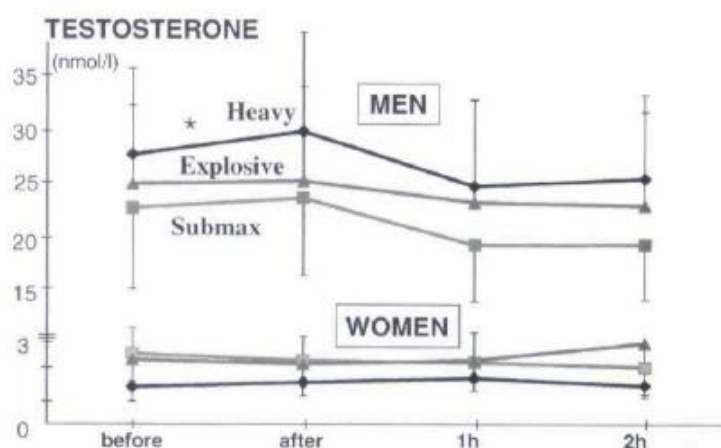


Fig. 9. Testosterone responses to different modes of resistance training in men and women. \*indicates significant different values compared to other protocols (Linnamo et al. 2005).

In addition, basal testosterone concentrations have been reported to be lower in women compared to men (Häkkinen & Pakarinen 1995) and hormonal changes in testosterone levels during heavy resistance training were higher in men whereas women maintained the same concentration during the loading and showed a slightly increase only during recovery (Linnamo et al. 2005).

Acute changes in cortisol concentrations seem to be higher in women compared to men. Almeida et al. (2009) found a 20% steeper awaking increase from nocturnal levels in women. During strength exercise, however, Häkkinen & Pakarinen (1995) found an increase of cortisol in men while women did not show any change in cortisol concentrations.

Caution must be paid when subject groups of women are analysed. Investigations including female subjects should be controlled for menstrual cycle based on consistent evidence that basal hormonal concentrations as well as acute changes in response to exercise are influenced by different cycle phases (Kraemer & Fleck 2004).

### 3 CHANGES IN PHYSIOLOGICAL VARIABLES AND FORCE PRODUCTION IN RESPONSE TO COMBINED ENDURANCE AND STRENGTH LOADINGS AND RECOVERY

Early investigations of Hickson (1980) demonstrated that the development of dynamic strength might be compromised by concurrent performance of both resistance and endurance training. Whereas prolonged combined training seems to negatively influence strength and power development (Bell et al. 1997), strength and power programs may be beneficial for endurance performance (Paavolainen et al. 1999). Investigations with regard to single session combined endurance and strength loadings are, however, lacking and in particular research dealing with the exercise order is, to the best of our knowledge, rare. The few available studies dealing with combined acute loading responses and chronic adaptations involved male subjects only. Data of female populations are currently rare.

#### 3.1 Influence of endurance loadings on subsequent force production

The phenomenon of strength inhibition during concurrent strength and endurance training has been associated with an acute hypothesis as introduced by Craig et al. (1991). It has been suggested that residual fatigue from the endurance loading component performed in the first half of a training session reduces the tension developed during the subsequent strength loading and, thus, leads to less effective strength development over time.

Table 3: Number of repetitions performed during each set of inertial squats in control conditions and following a strenuous endurance protocol (Leveritt & Abernethy 1999).

Number of set	Control (mean±SD)	Experimental (mean±SD)
Set 1	13.83±5.71	8.83±2.99
Set 2	11.17±4.45	8.17±3.60
Set 3	10.17±5.04	8.83±3.54

Indeed, the performance of endurance loadings considerable influences subsequent muscular force development. Following an intense bout of cycle intervals at intensities of 60%, 80% and 100% of  $VO_{2peak}$ , significant reductions in the number of sets performed in the subsequent strength sessions consisting of isoinertial and isokinetic exercises were observed (Table 3, Leveritt & Abernethy

1999).

As the blood lactate concentrations after the endurance protocol in the same study were significant higher compared to concentrations measured during the control trials, the authors of this study suggested that the related change in blood pH influences the quality of subsequent performed strength sessions. Denadai et al. (2007) further concluded that the strength loss after high intensity exhaustive running exercise (run to voluntary exhaustion at the lactate threshold) might be dependent on the contraction type and angular velocity of resistance protocols (Figure 11). According to the authors of the study the higher strength loss observed at the high angular velocity may be related to more pronounced muscular damage generated by eccentric contractions during the high intensity exercise.

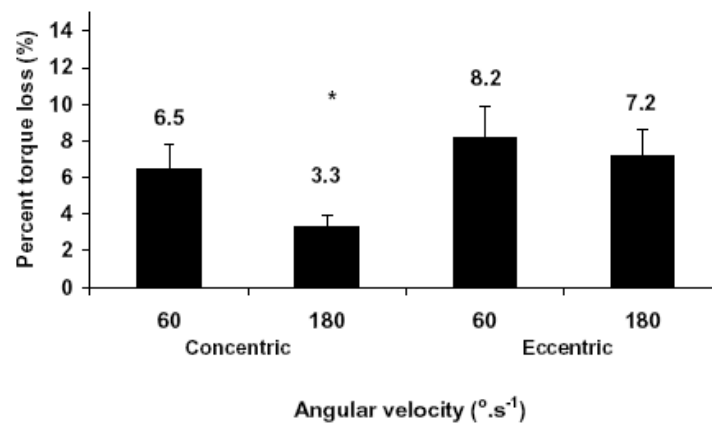


Fig. 10. Percent of concentric and eccentric peak torque loss at different angular velocities after intense endurance running. \* no significant change (Denadai et al. 2007).

De Souza et al. (2007) found a significant reduction in leg press repetitions performed with light strength-endurance like intensities following an intermittent high intensity interval training whereas a continuous run of lower intensity did not show any effects on strength performance. These findings clearly indicate the importance of endurance intensity and duration of subsequent inhibitions in strength performance.

It has been further suggested that the motor unit recruitment of strength-endurance type of resistance loadings are similar to that used in intense endurance running (De Souza et al. (2007). As in the same study no changes in upper body force production measured by the bench press were found, it can be suggested that acute reductions in strength performance initiated by endurance exercises are rather caused by neuromuscular mechanisms than metabolic fatigue as reflected by

lactic acid accumulation.

### 3.2 Influence of resistance exercises on subsequent endurance loadings

Single session combined strength and endurance loadings have recently been shown to impair endurance performance as indicated by running economy, when the endurance exercise is performed in the second half of the training session. According to Palmer & Sleivert (2001), running economy can even be impaired for up to eight hours following one single session of hypertrophy loadings (Figure 12). Moreover, it has been reported that the transient negative effects of resistance training on running economy can be incurred at slow as well as at faster sub-maximal running velocities.

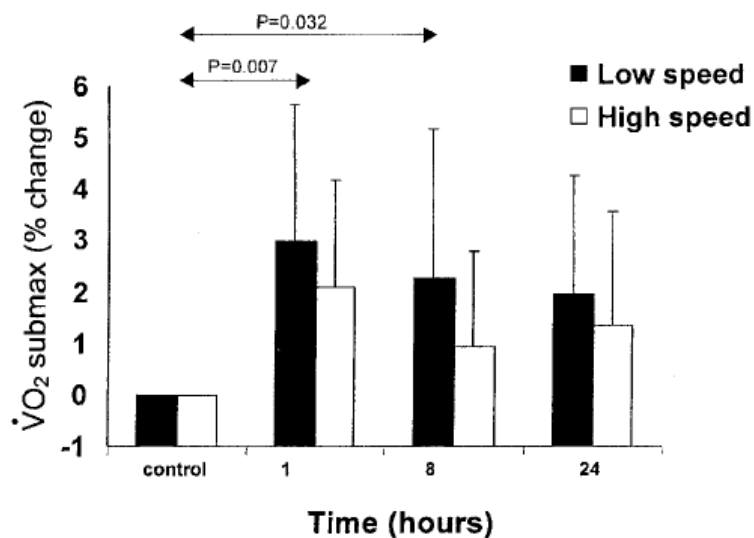


Fig. 11. Sub-maximal oxygen consumption during a 40min treadmill run expressed as % change relative to the control trial at low (mean±SD,  $13.4 \pm 2.3 \text{ km} \cdot \text{h}^{-1}$ ) and fast (mean±SD,  $14.7 \pm 2.3 \text{ km} \cdot \text{h}^{-1}$ ) velocity following a resistance training session (Palmer & Sleivert, 2001).

These findings are in accordance with reported results of Schuenke et al (2002) and Nagasawa (2008), which showed that post-exercise oxygen consumption was significantly increased following resistance exercises. It is reasonable to assume that increased rates of oxygen consumption above resting levels following a resistance loading session will considerably affect the metabolic demands and, thus, cause increases in running economy of the subsequent endurance run.

Mechanical efficiency has also been reported as an important determinant of running economy and any perturbation to this efficiency will subsequently increase aerobic demands (Anderson 1996).



Although in the study of Palmer & Sleivert (2001) no changes in stride length and frequency were measured, it remains possible that the whole body resistance workout may have altered biomechanical gait patterns which led to increased oxygen consumption.

In contrast to findings of Palmer and Sleivert (2001), however, Crawford et al. (1991) found no changes in aerobic demands during cycling at 65% following three sets of leg exercises at an intensity of 8RM. From this study it was concluded that resistance loadings restricted to the lower body only might not be sufficient to induce meaningful changes in work economy.

Marcora & Bosio (2007) investigated the influence of exercise induced muscle damage on endurance running performance and found a significant effect on endurance running performance (Figure 13). Subjects in this study performed an explosive strength protocol containing of 100 drop jumps followed by a 30min run to voluntary exhaustion. Although endurance performance was significantly decreased, no changes in physiological markers were found. In addition, running economy at 70%  $VO_{2max}$  was not affected. The authors of this study suggested that the decreased endurance performance was likely to be related to the sense of effort as running speed was lower and a significant correlation between rates of perceived exhaustion (RPE) was found.

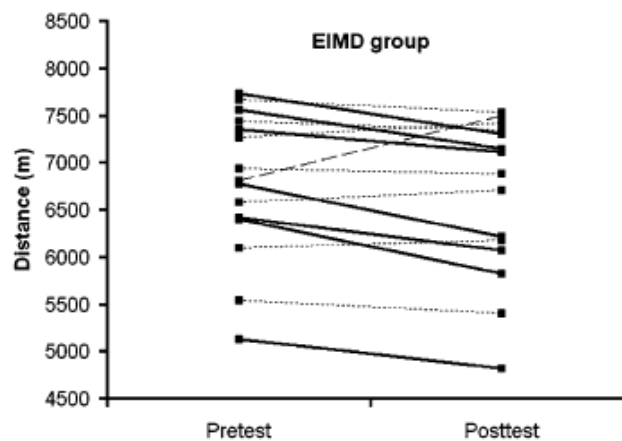


Fig. 12. Individual changes in time trial performance following intense an explosive strength protocol. Bold lines: decreases; Dashed lines: increases; Dotted line: no change (Marcora & Bosio, 2007).

### 3.3 Combined endurance and strength loadings and training

Most results with regard to combined endurance and strength loadings come from training studies conducted over prolonged times with combined endurance and strength loadings performed on

separate days. Some results are, however, available regarding the acute effects and chronic adaptations to single session combined endurance and strength loadings.

In a study of Brunetti et al. (2008) the influence of the exercise order on physiological variables was investigated and two different resistance loading sessions of 4x16RM and 4x8RM performed either before or after an aerobic endurance run at intensities of 60% and 80% of  $VO_{2max}$ . No changes were observed in blood lactate concentrations and oxygen consumption. Although the results may indicate that the order of single session combined endurance and strength loadings with regard to cardiovascular variables is not crucial, the research design may be criticized and the results should be interpreted with caution.

Chtara et al. (2005) investigated the chronic adaptations to single session combined endurance and strength training and found significant differences in endurance performance after 12 weeks of training (Table 4).

Table 4: Changes of physiological markers after 12 weeks of intra-session combined endurance and strength training (modified from Chtara et al. 2005)

Variable	Endurance + Strength	Strength + Endurance
4km trial	- 8.57%	- 4.66%
v $VO_{2max}$	+ 10.38%	+ 8.17%
$VO_{2max}$	+ 14.05%	+ 11.96%

In contrast to these findings of Chtara et al. (2005), in a study of Collins & Snow (1993) no order effect with regard to  $VO_{2max}$  was found and Gravelle & Blessing (2000) found increases in  $VO_{2max}$  to be compromised when an endurance loading preceded a subsequent resistance loading session in female subjects. Collectively these studies must be considered as precursors in the field of physiological responses to single session combined endurance and strength loadings and especially with regard to different exercise orders. Explanations for either of the findings remain, thus, speculative. It has been argued by the authors of these studies that accumulating fatigue may result in less than optimal performance of the exercise performed secondly in a combined loading session which may, in turn, inhibit chronic adaptations.

Similarly to changes in cardiorespiratory function, results with regard to chronic strength adaptations do not give evidence for an order effect when endurance and strength loadings are

combined in one single session (Chtara et al. 2008). In this study no significant difference in endurance strength, maximal strength and explosive power were observed when both loading conditions were compared. Both groups, however, improved less in maximal strength and explosive power when compared to a strength training only group indicating a possible interference between strength and endurance training.

Training studies of concurrent endurance and strength loadings performed on separate days commonly support these findings. It has been generally accepted that strength can be either compromised (Hennessy & Watson 1994) or unaffected (Sale et al. 1990). Hennessy and Watson (1994) reported that strength, power and speed performance may be most susceptible to interference because of the high intensity and volume of training performed. It needs to be, however, considered that most studies of concurrent endurance and strength training used non periodized programs over long periods of time. This is not always practical and might in particular indicate overtraining as an ultimate cause of exercise incompatibility (Fleck & Kraemer 2004).

More recently, growing evidence suggests that in contrast to strength inhibition endurance athletes may gain improvements in performance when including resistance sessions in their training program (Paavolainen et al. 1999; Laursen, 2005; Hamilton et al. 2006). Primarily explosive and maximum strength protocols have been shown to be beneficial by improving neuromuscular activation and reducing ground contact times and, thus, contributing to endurance performance improvements.

It has been shown that concurrent endurance and strength training alters the balance of anabolic to catabolic hormones which may reduce fibre hypertrophy and consequently inhibit strength development in both men and women (Leveritt & Abernethy 1999; Taipale et al. 2010). This was shown in a concurrent training study of Bell et al. (1997) who did not show significant differences in serum testosterone levels between strength training only and a combined group but increases in 1RM of leg press in strength training only group. As cortisol was significant increased in the combined group this might suggest a rather catabolic shift and may, thus, partly explain the suppressed strength development.

## 4 PURPOSE

The purpose of the present study was to examine acute changes in blood lactate concentrations and serum catabolic and anabolic hormone concentrations in response to combined strength (S) and endurance (E) loadings when performed in a single session (e.g. 1S+1E = single training session) in young, recreational endurance trained men and women. This study evaluated the “order effect” (E+S vs. S+E) and compared the differences in responses between men and women.

*Objective 1:* Prolonged endurance exercise is known to reduce a muscles’ ability to produce tension, and an acute response to a strength training session is a decrease in muscle activation and maximal and rapid force production. Strength loadings, on the other hand, have been shown to impair economy during a subsequent prolonged endurance session (Palmer & Sleivert 2001). Fatigue resulting from endurance or strength exercises performed first in a training session may affect the magnitude of decreases in neuromuscular and cardiorespiratory performance and efficiency during the second half of the training session. Changes in isometric voluntary force production of leg extensor muscles as in acute response to both loading conditions and recovery will, thus, be obtained. In addition, cardiovascular function indicated by changes in running economy will be determined during endurance running when this is performed both first and second in a single training session.

*Objective 2:* Both strength and endurance loadings have been shown to yield changes in blood lactate concentrations dependent on the exercise intensity in men and women. These findings have, however, likely been observed when both loadings were performed on separate days. Little is known regarding accumulation in blood lactate when both types of loading are performed in one single session. It will be, thus, determined whether the order of exercises has effects on the acute changes in blood lactate concentrations.

*Objective 3:* Strength and endurance exercises are known to induce specific acute responses in serum hormone concentrations. It remains, though, unclear how these responses differ when strength and endurance exercise are combined in one single session and if the acute changes are influenced by the order of exercises. Will performing endurance exercise first blunt acute endocrine responses to strength exercise in comparison to when strength exercise is performed first? The alterations within the endocrine system in acute responses and recovery to both exercise orders will

be comprehensively studied by analysing serum hormones from venous blood samples.

## 5 RESEARCH HYPOTHESIS

The hypothesis with regard to the research objectives were as follows:

1. Both maximal isometric force ( $MVC_{max}$ ) and the rapid portion of force production ( $MVC_{500}$ ) will be acutely decreased in men and women, independent of loading order. In addition, endurance performance as reflected by running economy will be impaired in both genders when endurance running is initiated by strength loadings.
2. The overall reduction in  $MVC_{max}$  and  $MVC_{500}$  will be of greater magnitude in men compared to women.
3. Decreased levels of  $MVC_{max}$  and  $MVC_{500}$  will return to baseline levels within 48 hours of recovery in men and women
4. Observed blood lactate concentrations will be generally of small magnitude in both men and women. Both strength and endurance loading will affect blood lactate concentrations of the subsequent loading in both genders and thus lead to increased accumulation.
5. Both loading conditions will cause increases in serum concentrations of anabolic and catabolic hormones independent of the loading order in both men and women.
6. Serum anabolic and catabolic hormone concentrations will return to baseline levels within 48 hours of recovery in both men and women.

## 6 METHODS

### 6.1 Subjects

A group of 10 female and 12 male subjects was recruited from the Jyväskylä region to participate in the study. All subjects were recreationally endurance trained with little or no experience in strength training. The subjects were free of acute and chronic illness, disease, injury or use of medications that would contraindicate participation in the study. The subjects were fully informed about the study design, including information on the possible risks prior signing an informed consent document. Health questionnaires and a resting ECG were reviewed by a qualified physician prior to testing. Ethical approval was granted by the University Ethical Committee, and the study was conducted according to the most recent Declaration of Helsinki.

The subject anthropometric data are shown in Table 5. In addition to standing height (wall-mounted tape measure, accuracy 0.1 cm) and subjects weight (digital scale, accuracy 0.1 kg), percentage of body fat was determined using skinfold calipometry (Durnin & Womersley 1974) and fat free mass was analyzed using bioimpedance (In body 720 body composition analyzer, Biospace CO. Ltd., Seoul, South Korea).

Table 5 Anthropometric data for female and male subjects obtained during the initial pre-testing.

Sex	Age [years]	Height [cm]	Weight [Kg]	Body fat [%]	FFM [Kg]	BMI
Female (N=10)	34±8	166.1±7.8	59.7±5.1	29.6±3.7	46.4±3.5	21.6±1.8
Male (N=12)	38±8	177.5±6.2	75.7±3.6	19.5±4.6	66.2±3.6	24.1±1.3

### 6.2 Design

The study was conducted within a cross over research design with all subjects participating in two loading conditions (Figure 14). Prior to the loadings subjects were tested for their maximal strength, power and endurance performance (basal measurements). Following these tests, subjects performed in a random order a loading of of strength and endurance combined in a single session. One loading session started with endurance loading immediately followed by strength loading(E+S) and one with the opposite order (S+E). The duration between the basal measurements and the first loading in

men and women was  $17 \pm 13$  days and  $22 \pm 17$  respectively. The duration between both loadings in men and women was  $25 \pm 16$  and  $20 \pm 14$  days, respectively. Time of day variations in neuromuscular and hormonal variables were controlled by the timing of each subjects loading  $\pm 1$  hour from their pre-testing time and by taking additional morning blood samples at rest on each testing day.

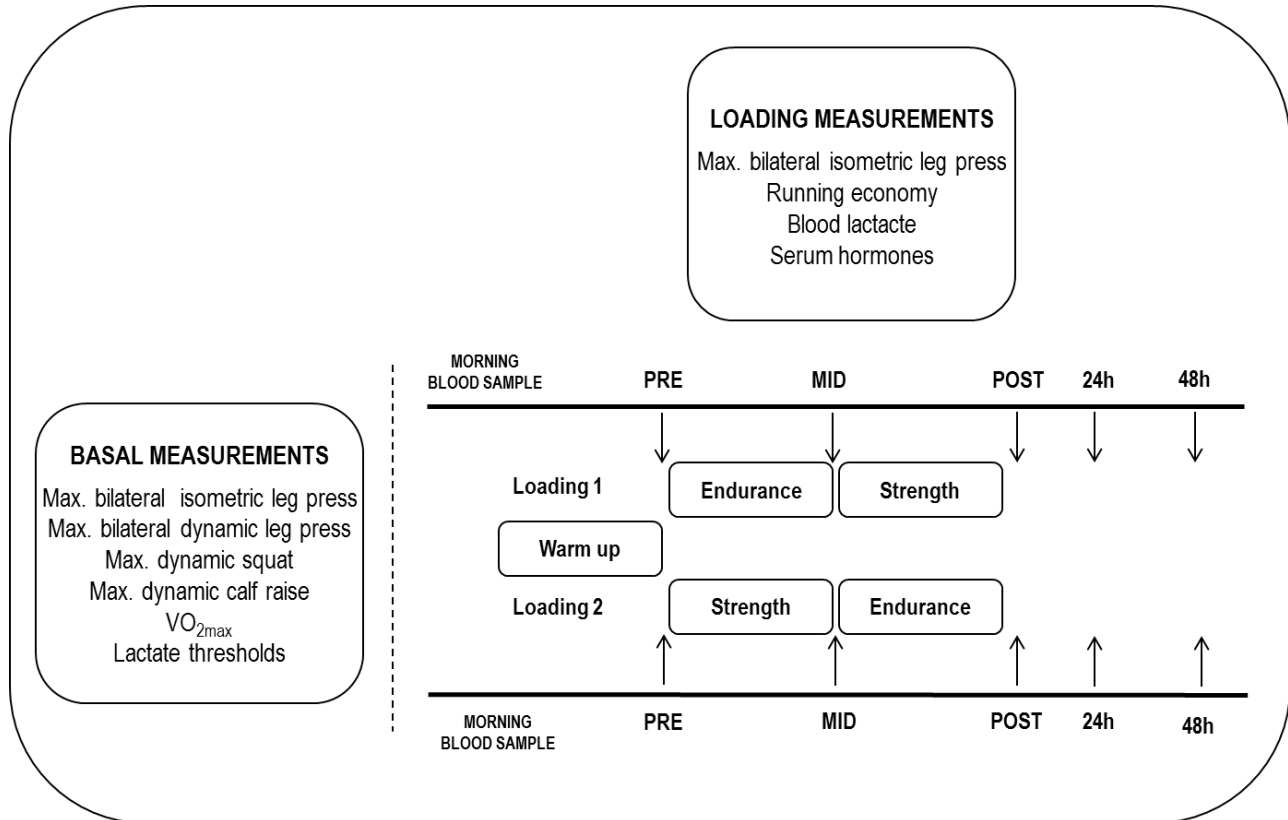


Fig. 13. Research design set up and measurements for both loading conditions.

Venous blood samples were drawn and strength and power measurements conducted prior the loading (PRE), immediately following endurance or strength exercises, respectively (MID) and after each combined session (POST). Follow up measures were conducted at 24h and 48h after each loading. Morning blood samples were drawn after 12h of fasting between 07:30 – 8:00 on the loading day and on follow up days at 24h and 48h to determine subjects' baseline endocrine profile and possible changes post recovery. In addition, blood lactate concentrations were determined from the fingertip at seven measurement times during each combined loading (Figure 15). The measurement times in both loading conditions were: pre strength loading, mid strength loading, post strength loading, pre endurance loading, at 10min and at 50min during the endurance loading and immediately post endurance loading. Subjects' body weight was measured before and after each combined loading in order to monitor sweating/hydration status.



### 6.3 Strength and Endurance loading

Both loading conditions (S+E vs. E+S) were started with a short warm up on a cycle ergometer with a duration of 5 minutes (Figure 15).

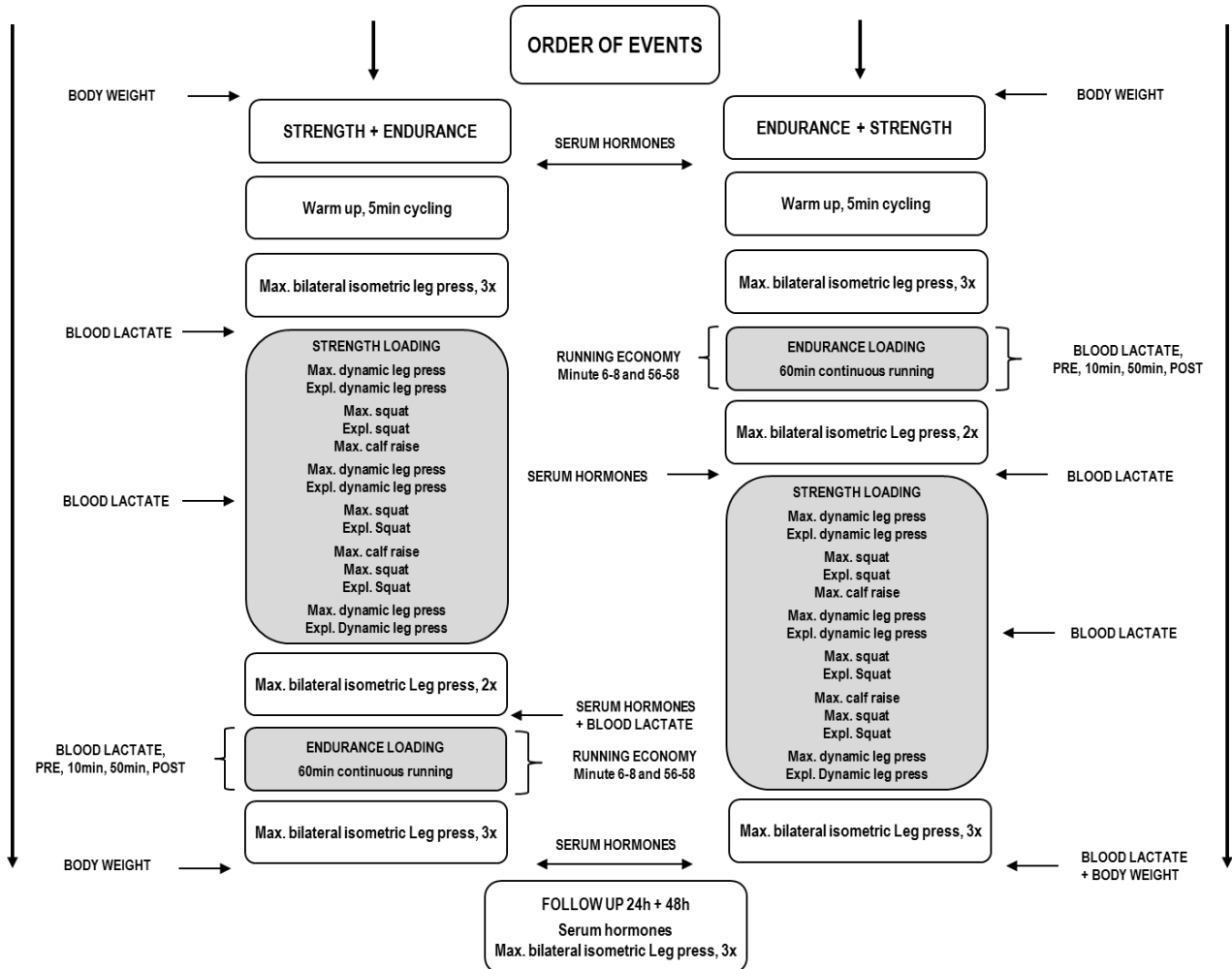


Fig. 15. Order of events in both E+S and S+E loadings.

*Strength loading.* The strength loading focused primarily on the leg extensor muscles and included both maximal strength and explosive power exercises with an overall duration of 40 minutes. Strength exercises were performed in a circuit such that leg press exercises were performed in the beginning, middle and end of the exercise session (Figure 15). Loads of 75% according to subjects determined 1RM load were used for maximal strength exercises which included two or three sets of 8 repetitions. The subjects were advised to perform a slow velocity over the whole range of movement and the final repetition of each set was performed near failure. Explosive power

exercises included three sets of 8-10 repetitions using 40% of subjects determined 1RM load. Exercises performed included: leg press (3 sets of maximal strength and 3 sets of explosive power), squats (3 sets of maximal strength and 3 sets of explosive power) and calf raises (2 sets of maximal strength). During maximal and explosive squats, subjects were allowed to wear a belt around their waist in order to stabilize their lower back. The rest between the sets was 2 minutes.

*Endurance loading:* The endurance loading was conducted with continuous running on a 200m indoor track. The intensity was a steady-state between each subjects' previously determined individual aerobic and anaerobic threshold for a duration of 60 minutes. The individual pace was given automatically by the light rabbit system integrated into the running track and was the same for both loading conditions. Heart rate, pace and distance run was measured constantly throughout the endurance exercise session in order to match both loading conditions with regard to work performed.

## 6.4 Measurements

*Basal measurements:* Prior to the loadings, subjects proceeded to the lab for the collection of anthropometric data and a specific set of tests including the measurement of maximal isometric leg extension ( $MVC_{max}$ ), rapid force production produced over 500ms ( $MVC_{500}$ ) and dynamic one repetition maximum (1RM). In addition, subjects were familiarized with the strength exercises performed in the following two loadings. Therefore, subjects four repetition maximum (4RM) in dynamic squat and calf raise was determined and based on that subjects probable 1RM estimated (Häkkinen & Ahtiainen 2007). Following the strength testing, maximal oxygen uptake ( $VO_{2max}$ ) was measured using a treadmill running protocol while aerobic and anaerobic thresholds were determined from gas analysis and blood lactates. The performed tests are described in detail below.

*One repetition maximum:* One repetition maximum (maximal dynamic bilateral horizontal leg press in a seated position) was measured using a David 210 dynamometer (David Sports Ltd., Helsinki, Finland). Prior to attempting 1RM, subjects completed a warm-up consisting of 6 x 70% RM and 4 x 80 – 85% 1 RM with one minute of rest between the sets. Following this warm up, no more than 5 attempts to reach 1 RM were made. Leg extension action in females and males started from an average knee angle of  $65.2 \pm 1.4$  and  $64.3 \pm 1.8$  degrees, respectively. Subjects were instructed to grasp the handles located by the seat of the dynamometer and to keep constant contact with the seat and backrest during leg extension to a full extension of 180 degrees. Verbal encouragement was

given to promote maximal effort. The greatest weight that the subject could successfully lift (knees fully extended) to the accuracy of 2.5 kg was accepted as 1RM.

*Isometric leg extension:* Maximal isometric bilateral leg extension force ( $MVC_{max}$ ) was measured on a horizontal dynamometer (Häkkinen et al. 1998) in a seated position with a knee angle of  $107^\circ$ . Subjects were instructed to generate maximum force as rapidly as possible through the entire foot against the force plate for a duration of 2 to 4 seconds. Subjects were asked to produce maximum force through the ball of the foot as rapidly as possible against the force plate for the same duration. In addition, subjects were instructed to grasp handles located by the seat of the dynamometer and to keep constant contact with the seat and backrest throughout each measurement trial. Verbal encouragement was given to promote maximal effort. In the pre-test subjects performed a minimum of 3 and a maximum of 5 trials with 1min rest in between. Maximal force was accepted when the difference between two subsequent trials did not exceed 5%. During the actual loading measurements, subjects performed 3 trials each at PRE, 24h and 48h with 1 min rest whereas at MID and POST only 2 subsequent trials without rest were performed. The force signal of both measures was low pass filtered (20 Hz) and analyzed (Signal software Version 4.04, Cambridge Electronic Design Ltd, Cambridge, UK). In addition, rapid force production of 500ms ( $MVC_{500}$ ) was measured during  $MVC_{max}$ .

*Dynamic squat and calf raises:* Subjects' 1RM in dynamic squat and calf raises was estimated from subjects performed 4RM. Squats and calf raises were determined using a Smith strength loading device with a tracked bar. For squat exercise subjects were asked to keep their feet shoulder width apart. During the eccentric phase subjects were instructed to bend their knees up to an angle between  $80^\circ$  and  $90^\circ$  and ensure that their knees did not bend forward beyond the level of the toes. For safety reasons subjects were allowed to use a hip-belt in order to support the lower back muscles. Prior attempting 4RM, subjects performed 2 to 3 sets of light weights in order to familiarize with the technique. After that, 4RM was approached with no more than 5 trials. However, if the subjects were able to exceed 4RM but reported lower back pain, a maximum of 6RM was accepted and based on that the estimated 1RM predicted. The greatest weight that the subject could successfully lift with the correct knee angle to the accuracy of 2.5 Kg was accepted as 4RM or 6RM, respectively.

*$VO_{2max}$  and blood lactate:* Maximal oxygen uptake ( $VO_{2max}$ ) and lactate thresholds were determined during treadmill running. The initial speed for men and women was  $8 \text{ km}\cdot\text{h}^{-1}$  and  $7 \text{ km}\cdot\text{h}^{-1}$ ,

respectively and it was increased by  $1 \text{ km}\cdot\text{h}^{-1}$  every third min until exhaustion. According to subjects training background the initial velocity was  $1 \text{ km}\cdot\text{h}^{-1}$  higher in subjects who reported a training background higher than the average. The incline for all subjects was kept at a constant  $0.5^\circ$  during the whole test. Heart rate was monitored continuously during the test (Suunto t6, Vantaa, Finland). Heart rate values used for analysis were means from the last min at each speed. Oxygen consumption was determined continuously breath-by-breath using a portable gas analyzer (Oxycon Mobile®, Jaeger, Hoechberg, Germany). Before each test, air flow calibration was performed using the automatic flow calibrator and the gas analyzer was calibrated against a certified gas mixture of 16 %  $\text{O}_2$  and 5 %  $\text{CO}_2$ . The  $\text{VO}_{2\text{max}}$  was taken as the highest 60-s  $\text{VO}_2$  value. Capillary blood samples were taken from the fingertip every 3<sup>rd</sup> min to measure blood lactate concentrations while the treadmill was stopped for about 15 – 20 sec. Running speed at the anaerobic threshold was determined from lactate and pulmonary variables using the same method as Aunola & Rusko (1986). Capillary blood samples of the initial treadmill test and both loadings were analyzed for blood lactate concentrations using Biosen lactate analyzer (S\_line Lab+, EKF Diagnostic, Magdeburg, Germany).

*Running economy and heart rate during endurance loadings:* Oxygen consumption ( $\text{VO}_2$ ) and running economy (RE) during the endurance loading was measured as the average of 3 minutes between minute 6 and 8 and minute 56 and 58, respectively. Oxygen consumption was determined continuously breath-by-breath using a portable gas analyzer (Oxycon Mobile®, Jaeger, Hoechberg, Germany). Before each test, air flow calibration was performed using the automatic flow calibrator and the gas analyzer was calibrated against a certified gas mixture of 16 %  $\text{O}_2$  and 5 %  $\text{CO}_2$ . Heart rate average was recorded throughout the whole endurance running duration (Suunto t6, Vantaa, Finland) but compared from minute 15 and 45 respectively.

*Serum hormones:* Venous blood samples (10ml) were collected using sterile needles into serum tubes (Venosafe, Terumo Mediacol Co., Leuven, Belgium) by a qualified lab technician. Whole blood was centrifuged at 3500rpm (Megafuge 1.0R, Heraeus, Germany) for 10min after which serum was removed and stored at  $-80^\circ\text{C}$  until analysis. Samples were used for determination of serum testosterone (TT), serum cortisol (C), serum growth hormone (GH) and serum sex hormone binding globulin (SHBG). Free testosterone was calculated according to the empirical free testosterone (FT) formula for high total Testosterone (TT) in men:

$$\text{High FT} = -52.65 + 24.4\text{TT} - 0.704\text{SHBG} - 0.0782\text{TT} \times \text{SHBG} - 0.0584\text{TT}^2$$

and low testosterone in women (Ly and Handelsmann 2005):

$$\text{Low FT} = -6.593 + 19.304\text{TT} + 0.056\text{SHBG} - 0.0959\text{TT} \times \text{SHBG}$$

Analyses were performed using chemical luminescence techniques (Immunlite 1.000) and hormone specific immunoassay kits (Siemens, New York, NY, USA). The sensitivity for serum hormones were: TT,  $0.5\text{nmol}\cdot\text{l}^{-1}$ ; C,  $5.5\text{nmol}\cdot\text{l}^{-1}$ ; GH,  $0.003\text{nmol}\cdot\text{l}^{-1}$  and SHBG,  $0.2\text{nmol}\cdot\text{l}^{-1}$ . The intra assay coefficients of variation were: TT, 8.7%; C, 7.1%; GH, 5.9% and SHBG, 6.5%.

## 6.5 Training and nutrition

Subjects were required to record their training volume and intensity for both strength and endurance training conducted in the period between both loadings. Subjects were asked to maintain their usual levels of physical activity throughout the whole period. However, not all subjects returned their training diaries. The analysis of available training records for women and men showed an average of  $3.7\pm 1.2\text{h}$  ( $n=7$ ) and  $4.2\pm 2\text{h}$  ( $n=9$ ), respectively. In addition, all subjects were asked to record their training conducted during three days prior to both loadings and were required not to perform any intensive exercises during this time.

In addition to subject's training, the nutritional intake was controlled on loading and follow up days. The subjects were required to ingest the same breakfast, similar in total amount and composition, on both loading and all follow up days. In addition to breakfast, subjects whose tests was conducted in the afternoon were asked to have a small snack about 2 hours prior the testing which was the same for both loadings and all follow up measures. During each loading, subjects were allowed to ingest a total amount of 4 dcl of pure tap water, with 2 dcl given separately right after the strength and after the endurance loading before the venous blood sample was drawn.

## 6.6 Statistical Analysis

Conventional statistical methods were used for the calculations of means and standard-deviation (SD). Between group differences (differences between men and women) were analysed using a one-way analysis of variance (Oneway ANOVA) as well as an independent-samples T-test. Within group differences were analysed using repeated measures ANOVA with 3 levels (PRE, MID, POST) and 5 levels (PRE, MID; POST, 24h, 48h) as well as a dependent-samples T-test (comparison of both loading conditions within one gender group). In the presence of a significant F-value, post-hoc

comparison of means was provided by Bonferroni's significance test. In addition, Pearson – product – moment correlation for serum hormones and strength variables was performed. The significance for all tests was set at \* $p < 0.05$ , \*\*  $p < 0.01$  and \*\*\* $p < 0.001$ . Statistical analysis was completed with PASW 18.0 (SPSS Inc., Chicago, IL, USA).

## 7 RESULTS

### 7.1 Basal measurements

Maximal heart rate of the basal treadmill test and blood lactate concentrations at the aerobic and anaerobic threshold were not statistically different between men and women, while maximal oxygen consumption as well as running speed at the aerobic and anaerobic threshold differed between the genders (Table 6).

Table 6. Treadmill data in men and women obtained during the initial pretesting. Values presented for maximal heart rate ( $HR_{max}$ ), maximal oxygen consumption ( $VO_{2max}$ ), blood lactate concentrations, running speed at the aerobic ( $AT_{lactate}$ ,  $AT_{speed}$ ) and anaerobic threshold ( $AnT_{lactate}$ ,  $AnT_{speed}$ ). \* $p < 0.05$ , \*\* $p < 0.01$  refers to significant differences compared to males.

Sex	$HR_{max}$ [bpm]	$VO_{2max}$ [ml·kg <sup>-1</sup> ·min <sup>-1</sup> ]	$AT_{lactate}$ [mmol·l <sup>-1</sup> ]	$AnT_{lactate}$ [mmol·l <sup>-1</sup> ]	$AT_{speed}$ [min·km <sup>-1</sup> ]	$AnT_{speed}$ [min·km <sup>-1</sup> ]
Female (n=10)	191±11	48.5±4.6**	1.4±0.3	3.2±0.5	06:29±0:30*	5:08±0:25**
Male (n=12)	185±10	47.9±4.8	1.6±0.4	3.2±0.7	05:50±0:33	04:36±0:22

All of the measured strength and power variables were significantly higher in men compared to women (Table 7).

Table 7. Maximal Strength values of the baseline measurements in men and women. Values are presented for one repetition maximum (1RM) in dynamic leg press (1RM), maximal bilateral leg extension ( $MVC_{max}$ ) and rapid force production over 500ms ( $MVC_{500}$ ). \*\*\* $p < 0.001$  refers to significant differences compared to males.

Sex	1RM [Kg]	$MVC_{max}$ [N]	$MVC_{500}$ [N]
Female (n=10)	103±21***	1954±441***	1233±302***
Male (n=12)	176±23	2920±487	1909±372

## 7.2 Endurance and strength performance

The running speed performed during the endurance loading was similar for both loading conditions in men (E+S, 04:57 min·km<sup>-1</sup> and S+E, 04:57 min·km<sup>-1</sup>) and in women (E+S, 05:38 min·km<sup>-1</sup> and S+E, 05:44 min·km<sup>-1</sup>). The speed in both loadings was significantly lower in women compared to men ( $p<0.01$ ). Oxygen consumption and heart rate during the endurance running in both loadings are shown in Table 8.

Table 8. Heart rate and oxygen consumption for both loading conditions in absolute values (mean±SD). Oxygen consumption is given as mean of minutes of 6-8 and 56-58, respectively. Heart rate values were obtained at minutes 15 and 45, respectively. \* $p<0.05$ , \*\* $p<0.01$ , \*\*\* $p<0.001$  refers to significant differences compared to the second corresponding value within each loading.

Sex	Loading S+E				Loading E+S			
	VO <sub>2</sub> [ml·kg <sup>-1</sup> ·min <sup>-1</sup> ]		Heart rate [bpm]		VO <sub>2</sub> [ml·kg <sup>-1</sup> ·min <sup>-1</sup> ]		Heart rate [bpm]	
	6' - 8'	56' - 58'	15'	45'	6' - 8'	56' - 58'	15'	45'
Female (n=10)	39.1±3.4	39.3±3.5	158±16	163±16**	37.9±6.1	38.5±3.8	155±16	161±15***
Male (n=12)	45.0±4.0	45.3±4.4	153±11	159±10***	43.3±3.7	44.7±9.6*	150±9	157±10***

The initial values of MVC<sub>max</sub> in both loadings were not different from values obtained during the pre-testing in both men and women, whereas MVC<sub>500</sub> was significantly reduced at PRE to both loadings (Table 9).

Table 9. Pre loading strength variables for both loading conditions in absolute values (mean±SD) for men and women. \*\*\* $p<0.001$  refers to significant differences compared to males.

Sex	Loading S+E		Loading E+S	
	MVC <sub>max</sub> [N]	MVC <sub>500</sub> [N]	MVC <sub>max</sub> [N]	MVC <sub>500</sub> [N]
Female (n=10)	1811±428***	1132±268***	1808±422***	1063±137***
Male (n=12)	2806±485	1812±421	2757±371	1836±324

Both men and women decreased their body weight significantly in S+E (women, 59.7±6.4 vs. 59.0±6.4,  $p<0.001$ ; men, 77.9±1.0 vs. 75.8±1.0 Kg,  $p<0.001$ ) and E+S (women, 59.9±4.9 vs. 59.2±4.8 Kg,  $p<0.001$ ; men, 76.2±6.1 vs. 74.7±5.7 Kg,  $p<0.001$ ).



### 7.3 Maximal and rapid isometric force production

Both loadings induced acute reductions in  $MVC_{max}$  in men at MID (E+S, 8%,  $p<0.05$ ; S+E, 19%,  $p<0.001$ ) and POST (E+S, 21%,  $p<0.001$ ; 19% S+E,  $p<0.001$ ), whereas in women only in S+E (14%,  $p<0.01$ ) at MID and in E+S (12%,  $p<0.01$ ) at POST (Figure 3).

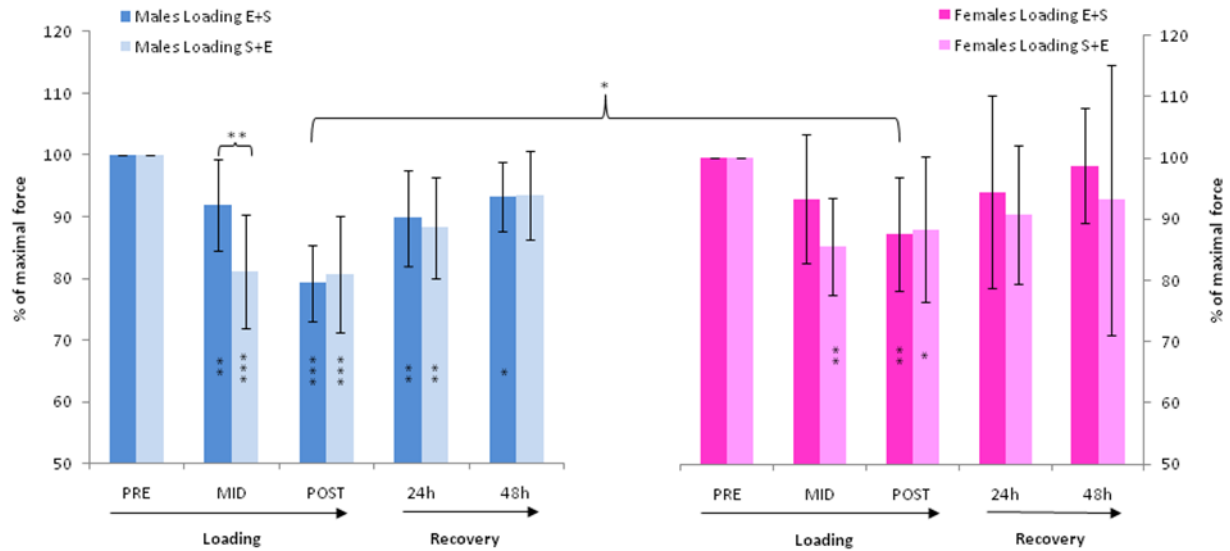


Fig. 16. Changes in bilateral isometric maximal force ( $MVC_{max}$ ) expressed relatively to PRE in both combined loadings for men and women in both loadings. \* $p<0.05$ , \*\* $p<0.01$ , \*\*\* $p<0.001$  refers to significant differences compared to PRE or as indicated.

The reduction of  $MVC_{max}$  in men at MID was higher following S+E compared to E+S (difference: 11%,  $p<0.01$ ).  $MVC_{max}$  remained reduced in men at 24h (E+S, 10%,  $p<0.01$ ; S+E, 12%,  $p<0.01$ ) and 48h (E+S, 7%,  $p<0.01$ ) but recovered in women.  $MVC_{500}$  decreased in men at mid (E+S, 14%,  $p<0.01$ ; S+E, 16%,  $p<0.05$ ) and post (E+S, 21%,  $p<0.01$ , S+E, 25%,  $p<0.001$ ) but not in women (Figure 17).

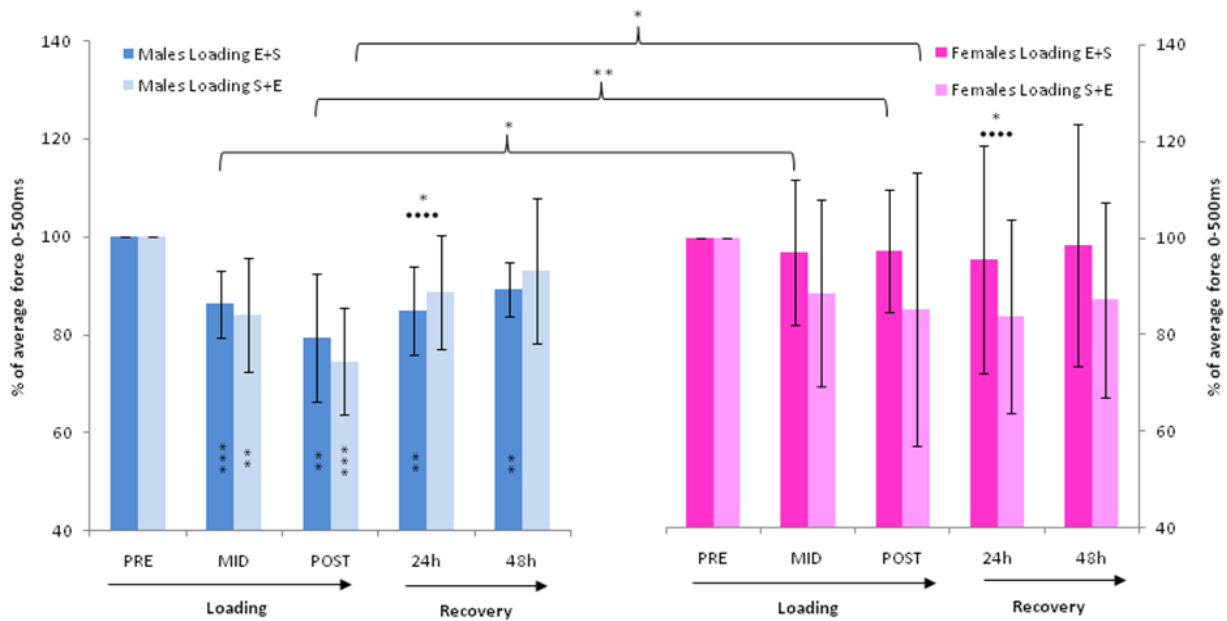


Fig. 17. Changes in rapid force production produced within 500ms during MVC (MVC500) expressed relatively to PRE in both combined loadings for men and women. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$  refers to significant differences compared to PRE or as indicated. \*\*\*\* refers to significant gender differences in the difference between E+S and S+E at the corresponding time point.

Although women did not show significantly reduced values in MVC<sub>500</sub> at 24h and 48h, the reduction following S+E was somewhat smaller compared to E+S and thus the difference of changes in MVC<sub>500</sub> between E+S and S+E in women at 24h was higher compared to the corresponding difference of men (-3% vs. +11%,  $p < 0.05$ ).

## 7.4 Blood lactate concentrations

Relative changes in blood lactate concentrations during the endurance running in men indicated an increase after 10min in E+S (>3 fold,  $p < 0.01$ ) and S+E (>2 fold,  $p < 0.01$ ) and in women only in S+E (<1 fold,  $p < 0.05$ ). The difference between E+S and S+E at the same time was higher in men compared to women ( $p < 0.05$ ). Post-endurance blood lactate concentrations were significantly increased in men in E+S (>2.5 fold,  $p < 0.05$ ) and women in S+E (>2 fold,  $p < 0.01$ ). Moreover, there was an order effect in relative changes in women at post ( $p < 0.05$ ) with higher increases observed in S+E compared to E+S. The absolute values of blood lactate concentrations obtained during the endurance loading are shown in Figure 18.

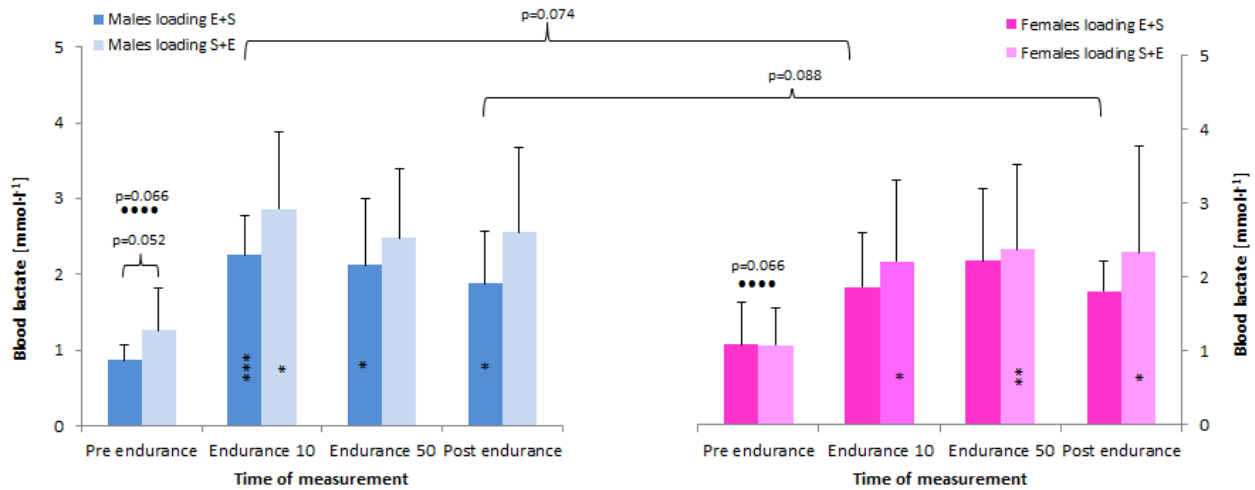


Fig. 18. Blood lactate concentrations as absolute values in men and women obtained during the endurance running in both combined loadings. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$  refers to significant differences compared to Pre-endurance or as indicated. ••• refers to significant gender differences in the difference between E+S and S+E at the corresponding time point.

Relative changes in blood lactate concentrations measured during the strength loading indicated an increase at Mid-strength in men in E+S (male, >2 fold,  $p < 0.05$ ) and S+E (male, > 2.5 fold,  $p < 0.01$ ) and in women only in S+E (>2.5 fold,  $p < 0.05$ ). At Post-strength the relative increase in men was maintained (E+S, <2 fold,  $p = 0.057$ ; S+E, >2 fold,  $p < 0.001$ ) and the difference between E+S and S+E was significant ( $p < 0.05$ ). Relative increases in blood lactate concentrations at Mid-strength and Post-strength were significantly higher in men compared to women (mid,  $p < 0.05$ ; post,  $p < 0.01$ ). The absolute values of blood lactate concentrations obtained during the strength loading are shown in Figure 19.

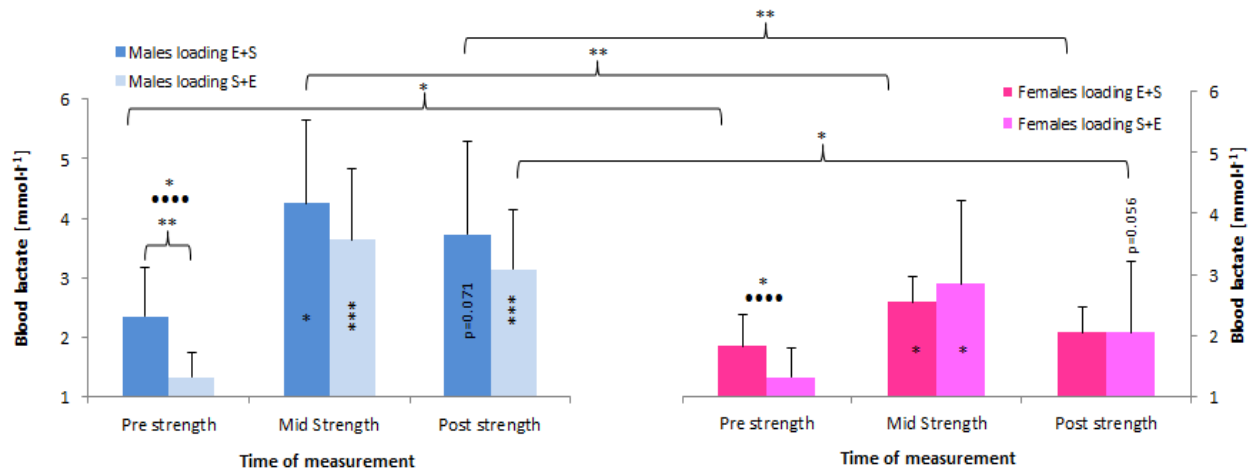


Fig. 19. Blood lactate concentrations as absolute values for men and women obtained during the strength loading in both combined loadings. \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$  refers to significant differences compared to Pre-strength or as indicated. \*\*\*\* refers to significant gender differences in the difference between E+S and S+E at the corresponding time point.

## 7.5 Running economy

Relative changes in RE in men indicated a significant increase in RE at Min 56-58 following E+S but not following S+E. No significant relative changes were found in women or between both genders. Absolute values of running economy are given in Figure 20.

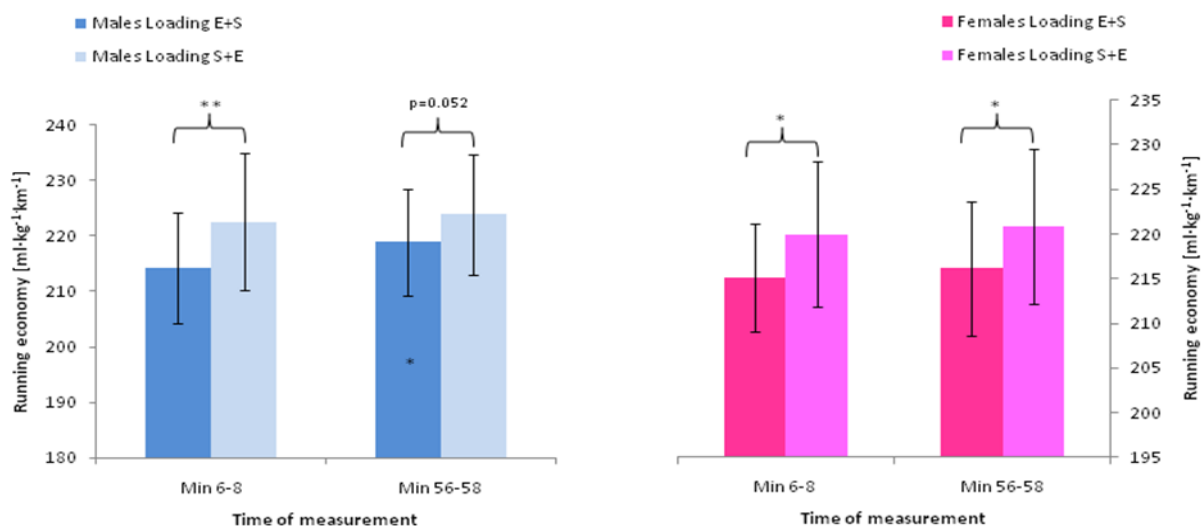


Fig. 20. Running economy as absolute values for men and women obtained during the endurance running in both combined loadings. \* $p < 0.05$ , \*\* $p < 0.01$  refers to significant differences compared to Min 6-8 or as indicated.

## 7.6 Serum hormone concentrations

Serum hormone concentrations of anabolic and catabolic hormones obtained in the morning of each loading and follow up day for women and men are presented in Table 10 and 11, respectively. Obtained concentrations of serum total testosterone in men in S+E at 24h were significant lower than the corresponding loading day value, while other serum hormone concentrations remained similar in men and women in both loading conditions.

Table 10. Absolute serum hormone concentrations obtained in the morning of each loading and follow up day in female subjects. Serum hormone concentrations shown for total testosterone (TT), free testosterone (FT) sex globuline binding protein (SHBG), growth hormones (GH) and cortisol (C) as mean±SD. It needs to be noted that n at follow up was only 6 and 5 for GH, respectively. \* refers to significant differences compared to the corresponding loading day value.

Serum hormone concentration	Loading S+E			Loading E+S		
	Loading	Follow up 24h	Follow up 48h	Loading	Follow up 24h	Follow up 48h
<b>TT</b> [nmol·l <sup>-1</sup> ]	1.12±0.44	1.02±0.20*	1.09±0.28	1.15±0.48	1.19±0.46	1.24±0.51
<b>FT</b> [nmol·l <sup>-1</sup> ]	0.01±0.01	0.01±0.00	0.01±0.00	0.01±0.01	0.01±0.00	0.01±0.01
<b>SHBG</b> [nmol·l <sup>-1</sup> ]	69.07±16.10	69.70±18.29	70.07±11.20	72.92±26.36	72.39±27.12	74.20±21.64
<b>GH</b> [mIU·l <sup>-1</sup> ]	4.68±5.19	2.27±2.96	2.68±2.68	8.84±13.21	3.04±6.55	1.80±1.49
<b>C</b> [nmol·l <sup>-1</sup> ]	449.00±62.32	428.78±61.17	477.33±64.37	477.67±70.44	453.33±88.86	385.67±128.32

Table 11. Absolute serum hormone concentrations obtained in the morning of each loading and follow up day in male subjects. Serum hormone concentrations shown for total testosterone (TT), free testosterone (FT) sex globuline binding protein (SHBG), growth hormones (GH) and cortisol (C) as mean±SD.

Serum hormone concentration	Loading S+E			Loading E+S		
	Loading	Follow up 24h	Follow up 48h	Loading	Follow up 24h	Follow up 48h
<b>TT</b> [nmol·l <sup>-1</sup> ]	15.84±2.94	13.79±2.36	14.51±5.07	15.96±4.24	15.47±3.35	15.72±4.05
<b>FT</b> [nmol·l <sup>-1</sup> ]	0.27±0.06	0.23±0.05	0.24±0.10	0.27±0.08	0.27±0.07	0.27±0.08
<b>SHBG</b> [nmol·l <sup>-1</sup> ]	35.09±8.14	34.85±7.82	34.94±8.79	34.65±10.08	34.04±8.48	35.66±8.96
<b>GH</b> [mIU·l <sup>-1</sup> ]	0.72±1.05	1.00±1.38	1.15±1.29	0.41±0.33	0.48±0.40	0.92±1.45
<b>C</b> [nmol·l <sup>-1</sup> ]	504.70±121.42	478.80±83.23	489.30±89.52	480.70±99.02	502.00±112.70	484.60±109.31

### 7.6.1 Acute responses in anabolic serum hormone concentrations

No significant acute changes occurred in serum total testosterone concentrations in either men or women (Figure 21). During recovery serum TT concentrations at 24h and 48h in men were slightly decreased (at 24h, 16%; at 48h, 8%) following S+E and slightly increased (at 24h, 7%; at 48h, 16%) following E+S at 24h (difference  $p<0.05$ ; at 48h, difference  $p<0.05$ ). No acute changes in free testosterone (FT) concentrations were observed. The difference at 24h and 48h recovery in FT was similar to the difference observed in TT (at 24h, difference  $p<0.05$ ; at 48h, difference  $p<0.05$ ). In both total and free serum testosterone concentrations obtained changes in S+E at POST and at 24h were higher in women compared to men (TT, POST  $p=0.084$ , 24h,  $p=0.068$ ; FT, POST,  $p=0.064$ ; 24h,  $p=0.061$ ).

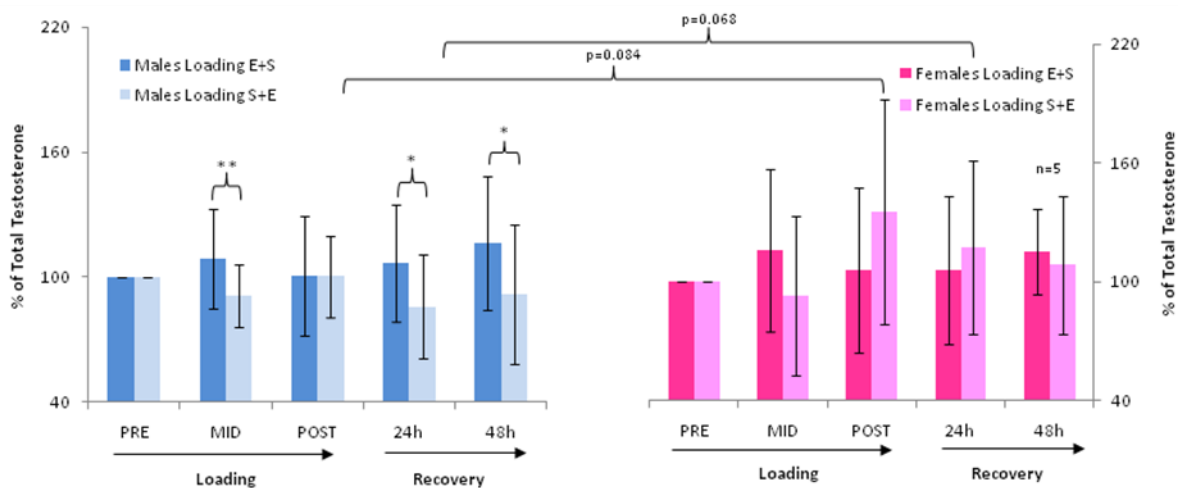


Fig. 21. Changes in total serum testosterone concentrations expressed relatively to PRE in both combined loadings for men and women. \* $p<0.05$  and \*\* $p<0.01$  refers to significant differences as indicated.

Serum SHBG concentrations were significantly increased in men at MID in E+S (5%,  $p<0.05$ ) and significantly reduced in S+E (6%,  $p<0.05$ ). The changes at MID were maintained at POST but appeared to be non-significant. In women no acute significant changes were observed at MID in both loading conditions but SHBG at POST was increased following S+E (10%,  $p<0.05$ ). The difference between E+S and S+E in women was significant ( $p<0.05$ ).

Serum GH concentrations were increased in men at MID in E+S (>75 fold,  $p<0.05$ ) and S+E (>70 fold,  $p<0.05$ ) and maintained at POST in S+E only (<60 fold,  $p<0.01$ ; Figure 22). The difference between E+S and S+E in men at POST was significant ( $p<0.05$ ). In women serum GH concentrations were slightly increased at MID following E+S and somewhat larger following S+E

( $p=0.085$ ; Figure 23). The difference between E+S and S+E between both genders at POST was significant ( $p<0.05$ ) with a larger difference observed in men. Serum GH concentrations at MID following E+S and POST following S+E were significantly larger in men compared to women ( $p<0.05$  and  $p<0.01$ , respectively). Serum GH concentrations returned to almost baseline at 24h in both men and women.

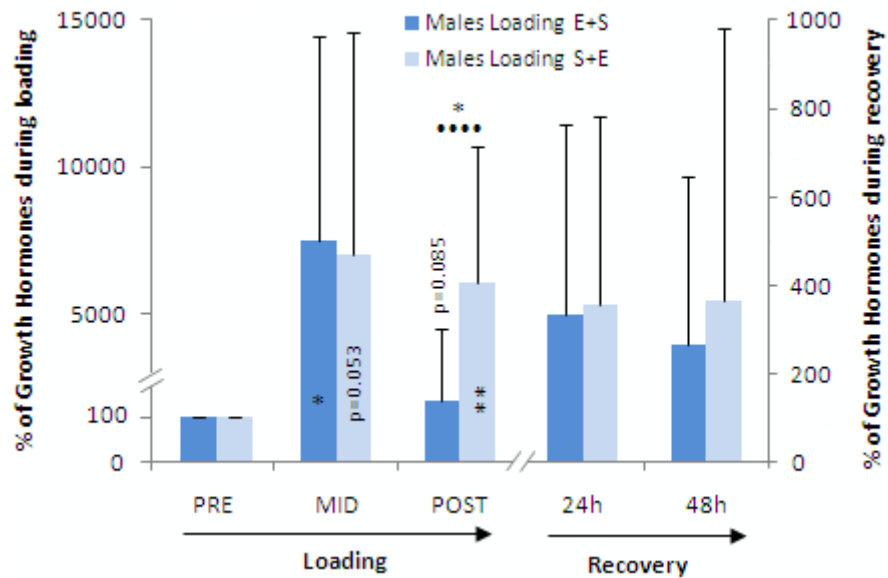


Fig. 22. Changes in serum GH concentrations expressed relatively to PRE in both combined loadings for men and women. \* $p<0.05$  and \*\* $p<0.01$  refers to significant differences compared to PRE, \*\*\* significant difference compared to females (Fig. 23) with regard to the difference between E+S and S+E at the corresponding time point.

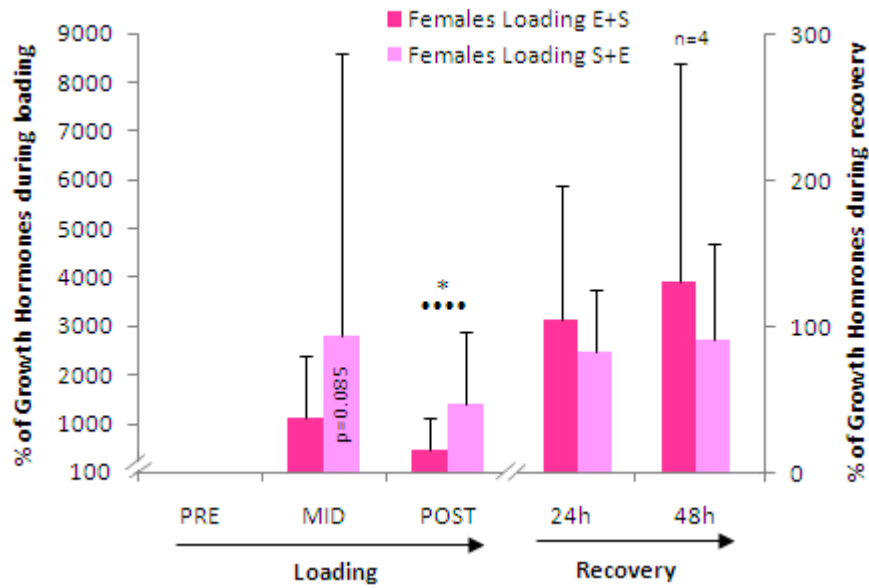


Fig. 23. Changes in serum GH concentrations expressed relatively to PRE (100%) in both combined loadings for men and women. \*\*\*\* significant difference compared to females (Fig. 22) with regard to the difference between E+S and S+E at the corresponding time point.

### 7.6.2 Acute responses in catabolic serum hormone concentrations

Serum cortisol concentrations were slightly increased in men at POST following S+E compared to E+S (46%,  $p=0.072$  vs. 1%). The difference between both loadings in men at POST was significant ( $p<0.05$ ). The increase in serum C concentrations in men at POST was higher ( $p<0.05$ ) compared to unaltered serum C concentrations in women (Fig. 24). In addition, serum C concentrations were reduced in women following both loading conditions at MID (E+S, 39%,  $p<0.01$ ; S+E (33%,  $p<0.01$ ) and POST following E+S (37%,  $p<0.05$ ).



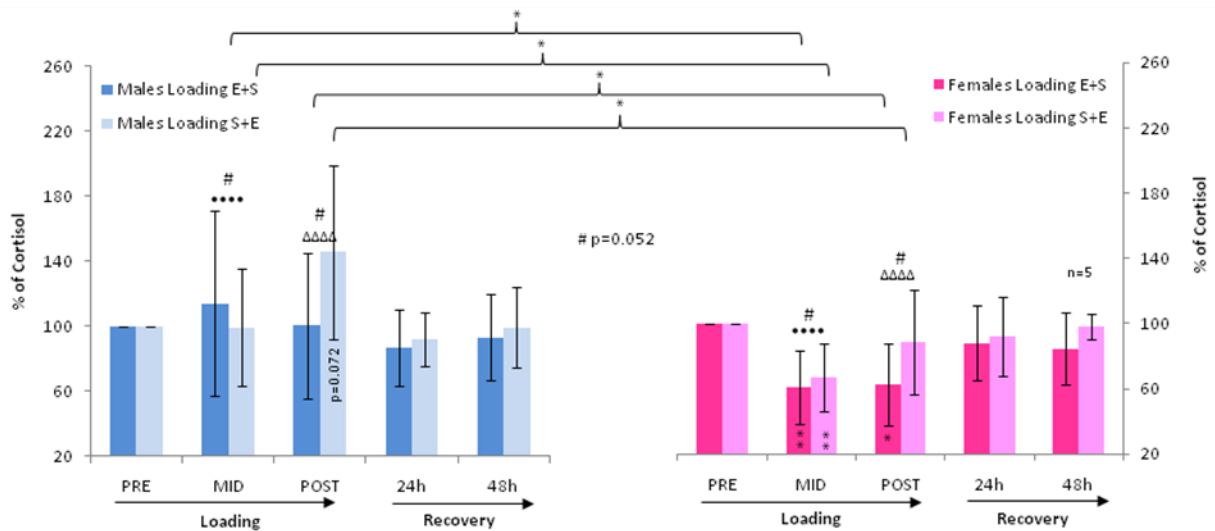


Fig. 24. Changes in serum cortisol concentrations relatively expressed to PRE in both combined loadings for men and women. \* $p < 0.05$  and \*\* $p < 0.01$  refers to significant differences compared to PRE or as indicated. •••• and  $\Delta\Delta\Delta\Delta$  refers to significance gender differences with regard to the difference between E+S and S+E at the corresponding time point.

## 7.7 Pearson-product-moment correlations

Changes in serum TT and FT concentrations were positively correlated with changes in serum GH concentrations in men at POST and 48h following E+S (TT: POST,  $r=0.619$ ,  $p=0.056$ ; 48h,  $r=0.676$ ,  $p < 0.05$ ; FT: POST,  $r=0.613$ ,  $p=0.059$ ; 48h,  $r=0.648$ ,  $p < 0.05$ ). Following S+E, changes in TT and FT were positively correlated with changes in serum GH concentrations in men at MID and 48h (TT: MID,  $r=0.827$ ,  $p < 0.01$ ; 48h,  $r=0.645$ ,  $p < 0.05$ ; FT: MID,  $r=0.839$ ,  $p < 0.01$ ; 48h,  $r=0.636$ ,  $p < 0.05$ ). No significant correlations between changes in serum TT and FT concentrations and GH concentrations were found in women.

## 8 DISCUSSION

The present study investigated acute changes in strength and endurance performance as well as responses of the endocrine system to single session combined endurance and strength loadings in recreationally trained male and female endurance runners. It was shown that both endurance and strength loadings performed first in a training session considerably influenced the performance of the subsequent loading and thus contributed to observed changes in physiological variables and force production. Most prominent were the findings of acute decreases in maximal isometric bilateral force production independent of the loading order in both men and women while these decreases were somewhat larger in men. Similarly, women did not show the same magnitude of reduction as men in rapid force production produced over 500ms in both E+S and S+E. From a physiological point of view, the current study showed that running economy in both genders was impaired throughout the endurance loading when this was performed immediately following the strength loading. Furthermore, higher serum cortisol concentrations immediately following S+E and decreased testosterone concentrations in men after S+E at 24h and 48h of recovery were observed.

### 8.1 Acute changes and recovery in maximal and rapid force production

Both strength and endurance loadings has previously been shown to lead to acute decreases in maximal voluntary force production and power. Typically, maximum strength loadings lead to acute fatigue in the neuromuscular system observed as decreases in both maximal voluntary neural activation and maximal force production of the exercised muscles (Häkkinen 1994).

The observed reductions in maximal isometric force production following S+E at MID were somewhat smaller as in previously reported results of Häkkinen (1993). Kraemer & Häkkinen (2002) indicated that the magnitude of fatigue induced acute changes in force production are related to the overall volume and intensity of the session and modified by the recovery between the sets. The loads used in the current study were, however, not as heavy as loads used in previous investigations and the rest period comparably long which might account for the smaller acute reductions in  $MVC_{max}$  found in the present study.

Following E+S, the observed reductions in maximal force production in both men and women when the endurance loading was performed first, were slightly smaller than those observed following

strength loading only. Previous research has shown that the magnitude of endurance exercise induced fatigue is specific to the intensity of endurance exercises performed and indicates that the current loading protocol caused moderate stress in both men and women (Gleeson et al. 1998).

The reductions in  $MVC_{500}$  observed in men and women following S+E at MID were of a smaller magnitude than those observed in  $MVC_{max}$ . Somewhat surprising was the finding that reductions in  $MVC_{500}$  at MID following E+S were slightly higher in men and slightly lower in women when compared to S+E. Häkkinen et al. (2003) stated that neuromuscular responses to endurance training occur on a smaller scale than those reported in resistance training because fewer motor units are activated during endurance exercise than in heavy resistance exercise. Whether this is true for acute responses remains, however, unclear. Reasons for decreases in performance of the rapid portion of the force production might be related to ultra structural changes and peripheral fatigue caused by prolonged performance of stretch shortening exercises during the endurance loading (Gleeson et al. 1998).

Although no order effect was found in men and women in both  $MVC_{max}$  and  $MVC_{500}$ , when analyzing both combined loading conditions it appears that higher reductions in  $MVC_{max}$  and  $MVC_{500}$  were caused by the strength loading rather than the endurance loading. Since the subjects were recreational trained endurance athletes with no or little experience in strength training, this finding was not a big surprise. Kramer and Häkkinen (2002) identified strength loading induced fatigue to be related to the training background of the subjects.

The current study did not show loading differences in the recovery at 24h and 48h in men and women in both  $MVC_{max}$  and  $MVC_{500}$ .  $MVC_{max}$ , however, was slightly more decreased in both men and women following S+E at 24h and in women only at 48h. Whereas in women this was true for  $MVC_{500}$  as well, the opposite trend was observed in men. Unfortunately, to date, no data are available regarding the acute recovery of single session combined endurance and strength loadings. Thus, reasons for the difference between both genders remain speculative but might be related to higher individual differences in women.

Of general importance was the finding that acute reductions in  $MVC_{max}$  and  $MVC_{500}$  following both loading conditions were somewhat higher in men compared to women and, thus, the recovery in women enhanced. Similarly, Häkkinen (1993) and Linnamo et al. (1998) found women to be less fatigable with regard to maximal and explosive strength. Although this observation is widely

accepted, the reasons are currently uncertain. According to Ryushi et al. (1988) and Häkkinen (1993), however, lower testosterone levels have been suggested which might cause a less rapid and aggressive activation of the exercised muscles. In addition, Fleck & Kraemer (2004) related lower magnitudes of fatigue in women to muscle fibre distributions. Heavy resistance loadings have been shown to rather activate fast twitch muscle fibres such as type IIb and IIa. However, these are less fatigue resistant compared to slow twitch type I fibres and as lower total numbers of type II muscle fibres have been observed in women this may explain the smaller rate of fatigue observed in females in the current study.

Collectively, these findings suggest that from a neuromuscular perspective and in particular with regard to strength performance the exercise order seems not to play an important role in either men or women. The results indicated that the exercise order becomes even less important for women as smaller magnitudes of fatigue and partly faster recovery during the subsequent days were observed.

## **8.2 Acute changes in blood lactate concentrations**

*Blood lactate concentrations obtained during the strength loading:* Accumulation of blood lactate concentrations with a resulting lowered blood pH have previously been shown to inhibit the rate of cross-bridge binding (Metzger & Moss 1990), leading to increased requirements for  $\text{Ca}_2^+$ , decreased maximal tension, decreased myosin adenosine triphosphatase activity and increased protein binding of  $\text{Ca}_2^+$  in the sarcoplasmic reticulum (Sahlin 1986). In particular, this might suggest blood lactate as a precursor of peripheral fatigue which in turn has dramatically effects on force production.

The observed blood lactate concentrations during the strength loading in the current study in both men and women, however, were of a small magnitude and thus cannot mainly account for reductions in force production. It has been reported that blood lactate concentrations are related to the type of loading performed. Whereas Häkkinen & Pakarinen (1993) showed that high volume loadings with long working periods per set leads to drastically increased blood lactate concentrations, Linnamo et al. (2005) and McCaulley et al. (2009) found explosive type of strength loadings to be insufficient to produce as high blood lactate concentrations. The resistance loading protocol in the current study contained of combined maximal and explosive type of strength exercises and blood lactate samples were drawn after the explosive set of strength exercise which might explain the comparably low blood lactate values observed.

A remarkably finding was, that the endurance loading led to increased blood lactate concentrations

in men at pre-strength in E+S but not in women indicated somewhat higher metabolic stress induced by the endurance run in men. Moreover, blood lactate at all measurement time points was higher in men compared to women and may support the hypothesis that women are less fatigable compared to men.

*Blood lactate concentrations obtained during the endurance loading:* The current study showed accumulated blood lactate during the endurance running in both men and women. Although not significant, both men and women showed higher blood lactate concentrations throughout the endurance run in S+E when compared to E+S.

The energy necessary to perform repeated sets of heavy resistance loadings is derived from ATP and phosphocreatine (PC) breakdown within the muscle. ATP, however, requires the breakdown of glucose molecules to pyruvate which causes blood lactate levels to rise (Henrikson 2000). In order to aerobically replenish ATP stores in the muscle, the oxygen consumption during the loading is increased and remains high immediately post loading for up to one hour (Hermansen et al. 1976). This might have influenced the oxygen consumption of the subsequent endurance run in the current investigation.

The gender difference in blood lactate concentrations observed during the strength loading became almost negligible during the endurance run. Explanations for that might lie in the muscle fibre distribution as stated above. During endurance exercises of low intensities performed over a prolonged duration, energy is mainly provided aerobically and, thus, slow twitch muscle fibres of type I are preferably recruited (Rush et al. 2000). The evident lack of fast twitch fibres (type IIb and IIa) in women becomes, therefore, negligible and blood lactate concentrations appear to be rather similar between both genders.

### **8.3 Acute changes in running economy**

When interpreting data of running economy, some caution must usually be exercised. Investigations of Morgan & Craib (1992) found the mean coefficient of variation in running economy as a result of 4 studies to range between 1 and 4%, which might limit the validity of these data. However, if, as in the present study, time of the day, footwear as well as training and performance activity are controlled, the coefficient of variation in recreational athletes was found to be as small as 1.32% in 68% of all trials and 2.64% in 95% of all trials (Morgan et al. 1990). Possible interference caused by external factors become, thus, negligible and the validity of the data is ensured.

The current findings of impaired running economy in both men and women following the strength loading were in accordance with results of Palmer & Sleivert (2001), who showed, that running economy might be even impaired for up to 8 hours at both low and high intensity endurance exercises.

To conclude underlying mechanisms for increased oxygen demands during subsequent endurance running in the current study is, however, difficult. It has previously been suggested that the pre-oxygen consumption ( $\text{VO}_2$ ) might have an influence on the oxygen kinetics during subsequent loadings (Sleivert & Palmer 2001). Pre-exercise  $\text{VO}_2$  was, however, not measured and thus conclusions cannot be drawn. In addition, increases in excess post-exercise oxygen consumption (EPOC) have been named as possible factors influencing the work economy of subsequent loading sessions (e.g. Schuenke et al. 2002) but have also not been measured in the current investigation.

Furthermore, it has been suggested that running economy is highly influenced by mechanical efficiency as represented in e.g. stride patterns of athletes (William & Cavanagh 1987). Certain stride variables were measured in the current study but their analysis has not yet been completed. Although decreases in maximal isometric leg extension give some evidence for central and/or peripheral fatigue, to date, no correlations between running mechanics and the increased oxygen demand caused by the strength loading have been performed.

In summary, the obtained findings in running economy have clearly shown that resistance loadings performed first in a training session considerably influences the oxygen demands of subsequent endurance loadings in both recreational trained men and women. Moreover, the magnitude of increases in running economy seems to be maintained throughout the endurance session.

#### **8.4 Acute changes and recovery of serum hormone concentrations**

*Serum testosterone and sex hormone binding globulin (SHBG) concentrations:* The present study showed, that neither of the loading protocols did show significant acute loading induced alterations in total and free testosterone in either men or women. At MID, however, serum total testosterone was slightly increased in E+S and slightly decreased in S+E in both men and women.

The finding that serum total testosterone responses were somewhat increased when obtained immediately after the endurance run performed first in the training session is in line with results of Daly et al. (2005) who found that testosterone concentrations during endurance loadings peak

between 60min and 120min. However, slightly decreased total and free testosterone responses at MID in S+E in both men and women, observed in the current study, are interesting since previous research showed rather increases or maintained values following intense bouts of resistance loadings (Häkkinen & Pakarinen 1995; Raastad et al. 2000). Reasons for the findings observed in the present study remain speculative. According to Schwab et al. (1993), a volume threshold might be needed to elicit significant increases in testosterone.

Furthermore, it remains unclear to which extent the training experience of the subject used in the current study affected acute testosterone responses. Previous results have indicated that serum testosterone concentrations in acute response to strength loadings are lower in untrained (Kraemer et al. 1998) and strength trained subjects (Gulledge et al. 1996; Hackney et al. 2003) when compared endurance athletes. The subjects in the current investigation were previously not experienced in resistance training but were recreationally involved in endurance training which might explain the lower obtained concentrations.

Serum total and free testosterone concentrations did not differ between loading conditions at POST in either of the gender groups. However, interesting was the finding that testosterone in men at 24h and 48h recovery was slightly increased following E+S and slightly decreased following S+E and the difference, thus, significant. This might suggest a prolonged recovery of the endocrine system in response to the S+E loading, indicated by excessive consumptions of testosterone by the muscles involved in the exercise.

A similar phenomenon was shown in a study of Häkkinen (1994), who found testosterone concentrations to be declined for up to 48 hours following an intense resistance exercise session whereas force production was already recovered. The authors of this study suggested that recovery of the neuromuscular and endocrine system takes place at different times with lower recovery rates of the endocrine system. In the current study, however, no significant decrease in testosterone but a significant difference between both loadings was observed in men. Whether this is related to delayed recovery from an endocrine perspective remains speculative.

The obtained serum total testosterone concentrations might be partly explained by obtained concentrations of sex hormone binding globulin (SHBG) which usually represents the bound portion of testosterone (Kraemer & Fleck 2004). SHBG in the current study showed similar responses as serum total testosterone which is in accordance with results of Häkkinen et al. (1985)

who found SHBG to reflect patterns of force production.

Collectively these findings suggest that neither the E+S nor the S+E loading protocol was stressful enough to yield significant alterations in serum total and free testosterone concentrations in recreationally endurance trained men and women. The data, however, might give some evidence for a delayed endocrine recovery process in men following S+E.

*Acute changes in serum growth hormone concentrations:* Serum growth hormone concentrations have previously been reported to be influenced by several external factors such as sleep, nutrition, training and alcohol consumption (Giustina & Veldhuis 1998). Furthermore, the same authors concluded that growth hormone release occurs in a pulsatile pattern with highest rates of secretion during the night. In the current study, however, factors such as nutrition and training were carefully controlled.

Previous research has shown that growth hormone concentrations are altered by both strength and endurance exercises whereas particularly in strength loadings the magnitude of changes seems to depend on the exercise selection, intensity and volume performed (Vanhelder et al. 1984; Kraemer et al. 1992). Growth hormone concentrations have also been shown to be altered in a similar manner as serum testosterone concentrations (Linnamo et al. 2005). This was supported by the current investigation as changes in serum total and free testosterone concentrations were positively correlated with serum growth hormone concentrations in men at POST and 48h 48 of recovery following E+S as well as following S+E at MID and 48h of recovery.

In addition, growth hormone concentrations have been reported to highly correlate with blood lactate concentrations and in particular  $H^+$  accumulation (Häkkinen & Pakarinen 1993). Even though blood lactate values obtained during both the strength and endurance loading in the current study were comparably low, increases in growth hormone responses were considerably increased and higher peak values as reported in previous studies were shown (Pritzlaff et al. 1999; Linnamo et al. 2005; Kraemer et al. 2008). According to Fleck & Kraemer (2004), however, other factors such as hypoxia, breath holding and protein catabolism may have an influence on growth hormone release and may explain the high changes in the current study at MID following both S+E and E+S.

Interestingly, the strength loading performed immediately after the endurance run did not show further increases in growth hormone concentrations but drastically reduced values in both men and



women. Of additional importance, might be the finding that the difference between both loadings at POST was higher in men compared to women. The presented results might suggest, that endurance running performed first in a training session may blunt growth hormone responses caused by strength exercises performed in the second half of the training session. To date it is, however, not possible to draw logical conclusions and further research is necessary to investigate possible mechanisms.

In summary, this study showed that single session combined endurance and strength loadings alter serum growth hormone concentrations in both men and women. To precisely explain the effect of exercise order on responses of the pituitary gland, however, further research is required.

*Acute changes in serum cortisol concentrations:* The observed increases in serum cortisol concentrations, particularly in men, reflect the overall loading stress induced by both the endurance and strength exercises. Most prominent was the finding of a significant trend of increased cortisol concentrations at POST following S+E in men.

Serum cortisol as a primary catabolic hormone has typically been associated with stress induced for example by injuries, surgeries or physical activity. According to a review by Smith (2000), resistance loadings in particular can be seen as adaptive trauma which leads to local acute inflammation, and ultimately to activation of the hypothalamic-pituitary-adrenal axis and thus rapid increases in serum cortisol concentrations. Similarly, cortisol has been shown to increase in response to prolonged endurance exercises (Virtanen et al. 1996; Tremblay et al. 2005). However, as with other hormones, previous data regarding the influence of the exercise order on cortisol concentrations are to our knowledge not available.

The prominent increase of serum cortisol in men at POST following S+E may indicate that this loading was somewhat more stressful than the loading with the opposite order (E+S). This was, however, not in line with obtained measures in maximal bilateral isometric leg extension since the increase in cortisol was positively correlated with reductions in maximal strength, indicating subjects who showed less fatigue in isometric strength showed higher concentrations in serum cortisol. Explanations for this finding are difficult to draw but the finding might reflect fatigue mechanisms of the neuromuscular and endocrine system independent of each other, as shown for recovery concentrations of testosterone (Häkkinen 1994).

Somewhat surprising was the finding that serum cortisol concentrations in women significantly decreased in both loading conditions at MID and remained decreased at POST. Cortisol has been shown to have a marked circadian rhythm. In a model of Rohatagi et al. (1996) it has been shown that cortisol decreases during the day in a linear pattern from approximately 4-5 a.m. to about midnight followed by a shorter linear increase of secretion from midnight to approximately 4-5 a.m. on the following day. It may be, thus, challenging to obtain accurate cortisol concentrations. Although all subjects in the current study performed both loadings at the same time of day, the time between the PRE blood sample and the actual start of the loading varied due to technical difficulties. It might be, thus, possible that time of the day influenced the obtained concentrations in serum cortisol.

It can be concluded that higher serum cortisol responses obtained in men at POST following S+E might indicate greater loading stress in this particular loading condition from an endocrinological point of view whereas in women the exercise order seems not to dramatically effect serum cortisol concentrations.

## **8.5 Limitations of the study**

Although this study provides some evidence for the order effect with regard to serum testosterone and cortisol responses in men, caution must be paid when assigning acute changes obtained in the current investigation to training adaptations over prolonged periods. Additional research is, thus, required to further explore to which extent acute disturbances in the homeostasis influences chronic adaptations to training over several weeks or month.

In addition, whether results of the current study obtained in recreational male and female endurance runners can be applied to other populations such as untrained subjects, elite athletes, strength athletes or elderly remains unclear. Further studies should therefore include different groups of subjects to verify findings observed in the current study.

## 9 CONCLUSIONS

The present study showed that single session combined endurance and strength loadings led to higher neuromuscular fatigue and larger responses of stress hormones in men than in women independent of the loading order. The current results provided some evidence to suggest that strength exercises followed by a subsequent endurance loading leads to higher loading induced stress in the endocrine system in recreational endurance trained men. Whereas the neuromuscular and endocrine stress was not reflected in metabolic blood lactate concentrations, the results suggested that running economy may be impaired in both men and women when endurance running is performed immediately following strength exercises. Collectively these findings might in turn have important implications to optimize the combined strength and endurance loading characteristics and its order as well as recovery from loading in recreationally endurance trained males and females.

## 10 REFERENCES

- AHTIAINEN, J.P., PAKARINEN, A., KRAEMER, W.J., & HAKKINEN, K. (2003a). Acute hormonal and neuromuscular responses and recovery to forced vs. maximum repetitions multiple resistance exercises. *International Journal of Sports Medicine*, **24**(6), 410-418.
- AHTIAINEN, J.P., PAKARINEN, A., ALEN, M., KRAEMER, W.J., & HAKKINEN, J. (2003b). Muscle hypertrophy, hormonal adaptations and strength development during strength training in strength-trained and untrained men. *European journal of applied physiology*, **89**(6), 555-563.
- ALMEIDA, D.M., PIAZZA, J.R., & STAWSKI, R.S. (2009). Interindividual differences and intraindividual variability in the cortisol awakening response: an examination of age and gender. *Psychology of Aging*, **24**(4), 819-827.
- ANDERSON, T. (1996). Biomechanics and running economy. *Sports Medicine*, **22**, 76–89.
- ASHLEIGH, J., McNICOL, A., BRENDAN, J., O'BRIEN, A., PATON, C.D., WADE, L., & KNEZ, C. (2009). The effects of increased absolute training intensity on adaptations to endurance exercise training. *Journal of science and medicine in sport*, **12**, 485-489.
- ÅSTRAND, P.O., HULTMAN, E., JUHLIN-DANNFELT, A., & REYNOLDS, G. (1986). Disposal of lactate during and after strenuous exercise in humans. *Journal of applied physiology*, **61**, 338-343.
- AUNOLA, S., & RUSKO, H. (1986). Aerobic and anaerobic thresholds determined from venous lactate or from ventilation and gas exchange in relation to muscle fiber composition. *International Journal of Sports Medicine*, **7**(3), 161-166
- BELL, G., SYROTUIK, D., SOCHA, T., MACLEAN, I., & QUINNEY, H.A. (1997). Effect of strength training and concurrent strength and endurance training on strength, testosterone, and cortisol. *Journal of Strength & Conditioning Research*, **11**(1), 57-64.
- BHASIN, S., WOODHOUSE, L, & STORER, T.W. (2001). Proof of the effect of testosterone on skeletal muscle. *Journal of Endocrinology*, **170**(1), 27-38.
- BORSHEIM, E., & BAHR, R. (2003). Effect of exercise intensity, duration and mode on post-exercise oxygen consumption. *Sports Medicine*, **33**(14), 1037-1060.
- BROOKS, S., NEVILL, M.E., MELEAGROS, L.; LAKOMY, HKA, HALL, G.M., BLOM, S.R., & WILLIAMS, C. (1990). The hormonal responses to repetitive brief maximal exercise in humans. *European Journal of Applied Physiology*, **60**, 144-148.
- BRUNETTI, A.P., ADOLFO, J., BRUM, P.P., SAMPAIO, V.M., DANTAS, E.H.M., & DOS SANTOS, M. Â. A. (2008). The Influence of the Order of Concurrent Training Session Over the Acute Response of the Blood Lactate, Heart Rate and Oxygen Uptake. *Fitness & Performance Journal (Online Edition)*, **7**(5), 326-331.
- CHICHARO, J.L., LOPEZ-CALDERON, A., HOYOS, J., MARTIN-VELASCO, A., VILLA, G., VILLANUA, M.A., & LUCIA A. (2001). Effects of an endurance cycling competition on resting serum insulin-like growth factor I (IGF-I) and its binding proteins IGFBP-1 and IGFBP-3. *British*

*Journal of Sports Medicine*, **35**(5), 303-307.

CHTARA, M., CHAMARI, K., CHAOUACHI, M., CHAOUACHI, A., KOUBAA, D., FEKI, Y., MILLET, G.P., & AMRI, M. (2005). Effects of intra-session concurrent endurance and strength training sequence on aerobic performance and capacity. *British journal of sports medicine*, **39**(8), 555-560.

CHTARA, M., CHAOUACHI, A., LEVIN, G.T. CHAOUACHI, M., CHAMARI, K., AMRI, M., & LAURSEN, P.B. E. (2008). Effect of concurrent endurance and circuit resistance-training sequence on muscular strength and power development. *Journal of Strength and Conditioning Research*, **22**, 1037-1045

COLLINS, M.A. and SNOW, T.K. (1993). Are adaptations to combined endurance and strength training affected by the sequence of training? *Journal of sports sciences*, **11**(6), 485-491.

CONSITT, L.A., COPELAND, J.L., & TREMBLAY, M.S. (2002). Endogenous anabolic hormone responses to endurance versus resistance exercise and training in women. *Sports Medicine*, **32**(1), 1-22.

CRAIG, B.W., LUCAS, J. POHLMAN, R., & STELLING, H. (1991). The effects of running, weightlifting and a combination of both on growth hormone release. *Journal of Applied Sport Scientific Research*, **5**, 198-203.

CRAWFORD, W.W., LOY, S.F., NELSON, A.G., CONLEE, R.K., FISHER, A.G. & ALLSEN, P.E. (1991). Effects of prior strength exercise on the heart rate oxygen uptake relationship during submaximal exercise. *Journal of Sports Medicine and Physical Fitness*, **31**(4), 505-509.

DALY, W., SEEGER, C.A., RUBIN, D.A., DOBRIDGE, J.D., & HACKNEY, A.C. (2005). Relationship between stress hormones and testosterone with prolonged endurance exercise. *European Journal of Applied Physiology*, **93**, 375-380.

DE SOUZA, E.O., TRICOLLI, V., FRANCHINI, E., PAULO, A.C., REGAZZINI, M., & UGRINOWITSCHI, C. (2007). Acute Effect of Two Aerobic Exercise Modes on Maximum Strength and Strength Endurance. *Journal of Strength & Conditioning Research*, **21**(4), 1286-1290.

GRATAS-DELAMARCHE, A., LE CAM, R., DELAMARCHE, P., MONNIER, M., & KOUBI, H. (1994). Lactate and catecholamine responses in male and female sprinters during a Wingate test. *European Journal of Applied Physiology*, **68**, 362-366.

DENADAI, B.S., GRECO, C.C., TUFIK, S., & DE MELLO, M.T. (2007). Effects of high intensity running to fatigue on isokinetic muscular strength in endurance athletes. *Isokinetics & Exercise Science*, **15**(4), 281-285.

DRUMMOND, M.J., VEHR, P.R., SCHAALJE, G.B., & PARCELL, A.C. (2005). Aerobic and Resistance Exercise Sequence Affects Excess Postexercise Oxygen Consumption. *Journal of Strength & Conditioning Research*, **19**(2), 332-337.

DURNIN, J.V., & WOMERSLEY, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *British Journal of Nutrition*, **32**(1), 77-97.

FILAIRE, E., DUCHE, P., & LAC, G. (1998). Effects of amount of training on the saliva

concentrations of cortisol, dehydroepiandrosterone and on the dehydroepiandrosterone: cortisol concentration ratio in women over 16 weeks of training. *European Journal of Applied Physiology & Occupational Physiology*, **78**(5), 466-471.

FLECK, S.J., & KRAEMER, W.J. (2004). Designing resistance training programs (3<sup>rd</sup> edition). Champaign, I.L: USA.

FLORINI, J.R., EWTON, D.Z. & COOLICAN, S.A. (1996). Growth hormone and the insulin-like growth factor system in myogenesis. *Endocrine reviews*, *17*, 481-517.

FRY, A.C., KRAEMER, W.J., STONE, M.H., WARREN, B.J., FLECK, S.J., KEARNEY, J.T., & GORDON, S.E. (1994). Endocrine responses to overreaching before and after 1 year of weightlifting. *Canadian Journal of Applied Physiology*, **19**, 400-410.

GAESSER, G.A., & BROOKS, G.A., (1984). Metabolic bases of excess post-exercise oxygen consumption: a review. *Medicine & Science in Sports & Exercise*, **16**(1), 29-43.

GILBERT, K.L., STROKES, K.A., HALL, G.M., & THOMPSON, D. (2008). Growth hormone responses to 3 different exercise bouts in 18- to 25- and 40- to 50-year-old men. *Applied Physiology, Nutrition & Metabolism*, **33**(4), 706-712.

GIUSTINA, A., & VELDHUIS, J.D. (1998). Pathophysiology of the neuroregulation of growth hormone secretion in experimental animals and the human. *Endocrine Reviews*, **19**, 717-797.

GLEESON, N.P., REILLY, T., MERCER, T.H., RAKOWSKI, S., & REES, D. (1998). Influence of acute endurance activity on leg neuromuscular and musculoskeletal performance. *Medicine & Science in Sports & Exercise*, **30**(4), 596 – 608.

GRANDYS, M., MAJERCZAK, J., DUDA, K., ZAPART-BUKOWSKA, J., KUIPA, J., & ZOLADZ, J.A. (2009). Endurance Training of Moderate Intensity Increases Testosterone Concentration in Young, Healthy Men. *International Journal of Sports Medicine*, **30**(7), 489-495.

GRAVELLE, B.L., & BLESSING, D.L. (2000). Physiological adaptation in women concurrently training for strength and endurance. *Journal of Strength and Conditioning Research*, *14*, 5-13.

GULLEDGE, T.P., & HACKNEY, A.C. (1996). Reproducibility of low resting testosterone concentrations in endurance trained men. *European Journal of Applied Physiology & Occupational Physiology*, **73**(6), 582-583.

GRIFFIN, J.E., & OJEDA, S.R. (2004). Textbook of endocrine physiology (5<sup>th</sup> edition). Oxford: University Press.

HACKNEY, A.C. (2008). Research methodology: Endocrinologic measurements in exercise science and sports medicine. *Journal of Athletic Training*, **43**(6), 631-639.

HACKNEY, A.C., SZCZEPANOWSKA, E., & VIRU, A.M. (2003). Basal testicular testosterone production in endurance-trained men is suppressed.. *European journal of applied physiology*, **89**(2), 198-201.

HÄKKINEN, K., PAKARINEN, A., ALEN, M., & KOMI, P.V. (1985). Serum hormones during prolonged training of neuromuscular performance. *European Journal of Applied Physiology & Occupational Physiology*, **53**(4), 287-293.

- HÄKKINEN, K. (1993). Neuromuscular fatigue and recovery in male and female athletes during heavy resistance exercise. *International Journal of Sports Medicine*, **14**, 53-59.
- HÄKKINEN, K., & PAKARINEN, A. (1991). Serum hormones in male strength athletes during intensive short term strength training. *European Journal of Applied Physiology*, **63**, 194-199.
- HÄKKINEN, K., & PAKARINEN, A. (1993). Muscle strength and serum testosterone, cortisol, and SHBG concentrations in middle-aged and elderly men and women. *Acta Physiologica Scandinavica*, **148**, 199-207.
- HÄKKINEN, K. (1994). Neuromuscular adaptation during strength training, ageing, detraining, and immobilization. *Critical Reviews in Physical and Rehabilitation Medicine*, **6**, 161-198.
- HÄKKINEN, K., & PAKARINEN, A. (1995). Acute hormonal responses to heavy resistance exercise in men and women at different ages. *International Journal of Sports Medicine*, **16**(8), 507-513.
- HÄKKINEN, K., NEWTON, R.U., GORDON, S.E., McCORMICK, M., VOLEK, J.S., NINDL, B.C., GOTSCHALK, L.A., CAMPBELL, W.W., EVANS, W.J., HÄKKINEN, A., HUMPHRIES, B.J., & KRAEMER, W.J. (1998). Changes in muscle morphology, electromyographic activity, and force production characteristics during progressive strength training in young and older men. *Journal of Gerontology: Biological Science*, **53**(6), 415-423.
- HÄKKINEN, K., ALEN, M., KRAEMER, W.J., GOROSTIAGA, E., IZQUIERDO, M., RUSKO, H., MIKKOLA, J., HÄKKINEN, A., VALKAINEN, H., KAARAKAINEN, E., ROMU, S., EROLA, V., AHTIAINEN, J., & PAAVOLAINEN, L. (2003). Neuromuscular adaptations during concurrent strength and endurance training versus strength training. *European Journal of Applied Physiology*, **89**(1), 42-52.
- HAMILTON, R.J., PATON, C.D., & HOPKINS, W.G. (2006). Effect of High-Intensity Resistance Training on Performance of Competitive Distance Runners. *International Journal of Sports Physiology & Performance*, **1**(1), 40-49.
- HAWLEY, J.A. (2009). Molecular responses to strength and endurance training: Are they incompatible? *Applied Physiology, Nutrition & Metabolism*, **34**(3), 355-361.
- HAWLEY, J.A. (2002). Adaptations of skeletal muscle to prolonged, intense endurance training. *Clinical & Experimental Pharmacology & Physiology*, **29**(3), 218-222.
- HENNESSY, L.C., & WATSON, A.W.S. (1994). The interference effects of training for strength and endurance simultaneously. *Journal of Strength and Conditioning Research*, **8**, 12-19.
- HENRIKSSON (2000). Cellular metabolism and endurance, edited by R.J. SHEPHARD & P.-O. ÅSTRAND, 84-102. London: Blackwell Science Ltd.
- HERMANSEN, L., MACHLUM, S., PRUETT, E.R., VAAGE, O., WALDRUM, H., & WESSEL-AAS, T. (1976). Lactate removal at rest and during exercise. In *Metabolic adaptation to prolonged physical exercise*, edited by H. HOWARD & J.R. POOTSMANS, 101-105. Basel: Birkhäuser Verlag.
- HICKSON, R.C. (1980). Interference of strength development by simultaneously training for strength and endurance. *European Journal of Applied Physiology & Occupational Physiology*, **45**(2), 255-263.

HOFF, J., GRAN, A., & HELGRUD, J. (2002). Maximal strength training improves aerobic endurance performance. *Scandinavian Journal of Medicine and Science in Sports*, **12**(5), 288-295.

INGJER, F. (1969). Effects of endurance training on muscle fibre ATP-ase activity, capillary supply and mitochondrial content in men. *Journal of Physiology*, **294**, 419-432.

KANG, J., HOFFMAN, J.R., IM, J., SPIERING, B.A., RATAMESS, N.A., RUNDELL, K.W., NIOKA, S., COOPER, J., & CHANCE, B. (2005). Evaluation of Physiological Responses during Recovery Following Three Resistance Exercise Programs. *Journal of Strength & Conditioning Research (Allen Press Publishing Services Inc.)*, **19**(2), 305-309.

KARKOULIAS, K., HABEOS, I., CHAROKOPOS, N., TSIAMITA, M., MAZARAKIS, A., POULI, A., & SPIROPOULOS, K. (2008). Hormonal responses to marathon running in non-elite athletes. *European Journal of Internal Medicine*, **19**, 598-601.

KEIZER, H.A. (1998). Neuroendocrine aspects of overtraining. In: KREIDER, R.B., FRY, A.C. and O'TOOLE, M.L., *Overtraining in sport*. Champaign (IL): Human Kinetics.

KOKALAS, N., TSALIS, G., & TSIGILIS, N. (2004). Hormonal responses to three training protocols in rowing. *European Journal of Applied Physiology*, **92**, 128-132.

KRAEMER, W.J., GORDON, S.E., FLECK, S.J., MARCHITELLIS, L.J., MELLO, R., DZIADOS, K., HARMAN, E., MARESH, C., & FRY, A.C. (1991). Endogenous anabolic hormonal and growth factor responses to heavy resistance exercise in males and females. *International Journal of Sports Medicine*, **12**(2), 229-235.

KRAEMER, W.J., FRY, A.C., WARREN, B.J., STONE, M.H., FLECK, S.J., KEARNEY, J.T., CONROY, B.P., MARESH, C.M., WESEMAN, C.A., TRIPLETT, N.T., & GORDON, S.E. (1992). Acute hormonal responses of elite junior weightlifters. *International Journal of Sports Medicine*, **12**, 228-235.

KRAEMER, W.J., PATTON, J.F., GORDON, S.E., HARMAN, E.A., DESCHENES, M.R., REYNOLDS, K., NEWTON, R.U., TRIPLETT, N.T., & DZIADOS, J.E. (1995). Compatibility of high-intensity strength and endurance training on hormonal and skeletal muscle adaptations. *Journal of Applied Physiology*, **78**(3), 976-989.

KRAEMER, W.J., STARON, R.S., HAGERMAN, F.C., HIKIDA, R.S., FRY, A.C., GORDON, S.E., NINDL, B.C., GOTSCHALK, L.A., VOLEK, J.S., MARX, J.O., NEWTON, R.U., & HÄKKINEN, K. (1998). The effects of short term resistance training on endocrine function in men and women. *European Journal of Applied Physiology*, **78**, 69-76.

KRAEMER, W.J., HÄKKINEN, K., NEWTON, R.U., NINDL, B.C., VOLEK, J.S., McCORMICK, M., GOTSCHALK, L.A., GORDON, S.E., FLECK, S.J., CAMPBELL, W.W., PUTUKIAN, M., & EVANS, W.J. (1999). Effects of heavy-resistance training on hormonal response patterns in younger vs. older men. *Journal of Applied Physiology*, **87**(3), 982-992.

KRAEMER, W.J., HARMAN, F.S., VOS, N.H., GORDON, S.E., NINDL, B.C., MARX, J.O., GOMEZ, A.L., VOLEK, J.S., RATAMESS, N.A., MAZETTI, S.A., BUSH, J.A., DOHI, K., NEWTON, R.U., & HÄKKINEN, K. (2000). Effects of exercise and alkalosis on serum insulin-like growth factor I and IGF-binding protein-3. *Canadian Journal of Applied Physiology*, **25**(2), 127-137.



KRAEMER, W.J., & HÄKKINEN, K. (2002). *Strength training for sport*. Blackwell Science Ltd: New York.

KRAEMER, W.J., FRAGALA, M.S., WATSON, G., VOLEK, J.S., RUBIN, M.R., FRENCH, D.N., MARESH, C.M., VINGREN, J.L., HATFIELD, D.L., SPIERING, B.A., YU-HO, J., HUGHES, S.L., CASE, H.S., STUEMPFLE, K.J., LEHMANN, D.R., BAILEY, S., & EVANS, D.S. (2008). Hormonal responses to a 160-km race across frozen Alaska. *British journal of sports medicine*, **42**(2), 116-120.

KRAEMER, W.J., & RATAMESS, N.A. (2004). Fundamentals of resistance training: progression and exercise prescription. *Medicine & Science in Sports & Exercise*, **36**(4), 574-688.

KUOPPOSALMI, (1981). Effects of exercise stress on human plasma hormone levels – with special reference to steroid hormones. Helsinki: Publishing Unit, University Library of Helsinki.

KUOPPOSALMI, K., NÄVERI, H., HÄRKÖNEN, M., & ADLERCREUTZ, H. (1980). Plasma cortisol, androstenedione, testosterone and luteinizing hormone in running exercise of different intensities. *Scandinavian Journal of clinical laboratory investigations*, **40**, 403-409.

LAURSEN, P.B., CHISWELL, S.E., & CALLAGHAN, J.A. (2005). Should Endurance Athletes Supplement Their Training Program With Resistance Training to Improve Performance? *Strength & Conditioning Journal (Allen Press)*, **27**(5), 50-55.

LEVERITT, M., & ABERNETHY, P.J. (1999). Acute effects of high-intensity endurance exercise on subsequent resistance activity. *Journal of Strength & Conditioning Research*, **13**(1), 47-51.

LINNAMO, V., HÄKKINEN, K., & KOMI, P.V. (1998). Neuromuscular fatigue and recovery in maximal compared to explosive strength loading. *European Journal of Applied Physiology*, **77**, 176-181.

LINNAMO, V., PAKARINEN, A., KOMI, P.V., KRAEMER, W.J., & HÄKKINEN, K. (2005). Acute Hormonal Responses to Submaximal and Maximal Heavy Resistance and Explosive Exercises in Men and Women. *Journal of Strength & Conditioning Research (Allen Press Publishing Services Inc.)*, **19**(3), 566-571.

LY, L.P., & HANDELSMAN, D.J. (2005). Empirical estimation of free testosterone from testosterone and sex hormone-binding globulin immunoassays. *European Journal of Endocrinology*, **152**, 471-478.

MANETTA, J., BRUN, J.F., MAIMOUN, L., FEDOU, C., PREFAUT, C., & MERCIER, J. (2003). The effects of intensive training on insulin-like growth factor 1 (IGF-1) and IGF binding proteins 1 and 3 in competitive cyclists: relationship with glucose disposal. *Journal of Sports Sciences*, **21**(3), 147-154.

McCRAE, H., DENNIS, S., BOSCH, A., & NOAKES, T. (1992). Effects of training on lactate production and removal during progressive exercise in humans. *Journal of applied physiology*, **72**(5), 1649-1656.

MARCORA, S.M., & BOSIO, A. (2007). Effect of exercise-induced muscle damage on endurance running performance in humans. *Scandinavian journal of medicine & science in sports*, **17**(6), 662-671.

- MARIEB, E.N. (2004). Human anatomy & physiology (6<sup>th</sup> edition). San Francisco: Pearson Benjamin Cummings.
- MCARDLE, W.D., KATCH, F.I. and KATCH, V.L., (2007). Exercise physiology : energy, nutrition, and human performance. (6th edition). Philadelphia (Pa.): Lippincott Williams & Wilkins.
- MCCAULLEY, G.O., MCBRIDE, J.M., CORMIE, P., HUDSON, M.B., NUZZO, J.L., QUINDRY, J.C., & TRIPLETT, N.T. (2009). Acute hormonal and neuromuscular responses to hypertrophy, strength and power type resistance exercise. *European journal of applied physiology*, **105**(5), 695-704.
- MCNICOL, A.J., O'BRIEN, B.J., PATON, C.D., & KNEZ, W.L. (2009). The effects of increased absolute training intensity on adaptations to endurance exercise training. *Journal of Science & Medicine in Sport*, **12**(4), 485-489.
- METZGER, J., & MOSS, R. (1990). pH modulation of the kinetics of Ca<sup>2+</sup> sensitive cross-bridge state transition in mammalian single skeletal muscle fibers. *Journal of Physiology*, **428**, 751-764.
- MIKKOLA, J.S., RUSKO, H.K., NUMMELA, A.T., PAAVOLAINEN, L.M., & Häkkinen, K. (2007). Concurrent endurance and explosive type strength training increases activation and fast force production of leg extensor muscles in endurance trained athletes. *Journal of Strength and Conditioning Research*, **21**(2), 613-620.
- MORGAN, D.W., MARTIN, P.E., BALDINI, F.D., & KRAHENBUHL, G.S. (1990). Effects of a prolonged maximal run on running economy and running mechanics. *Medicine and Science in Sport and Exercise*, **22**(6), 834-840.
- MORGAN, D. W., & CRAIB, M. (1992). Physiological aspects of running economy. *Medicine and Science in Sport and Exercise*, (24), 456-461.
- NAGASAWA, T. (2008). Resistance exercise increases postexercise oxygen consumption in nonexercising muscle. *European journal of applied physiology*, **104**(6), 1053-1059.
- NÄVERI, H, KUOPASALMI, K., & HÄRKÖNEN, M. (1985). Metabolic and hormonal changes in moderate and intense long-term running exercises. *International journal of sports medicine*, **6**, 276-281.
- NEVILL, M.E., HOLMYARD, D.J., HALL, G.M., ALLSOP, P., VAN OOSTERHOUT, A., BURRIN, J.M., & NEVILL, A.M. (1996). Growth hormone responses to treadmill sprinting in sprint- and endurance-trained athletes. *European Journal of Applied Physiology & Occupational Physiology*, **72**(5), 460-467.
- NGUYEN, U.N., MOUGIN, F., SIMON-RIGAUD, M., ROUILLON, J.D., MARGUET, P., & REGNARD, J. (1998). Influence of exercise duration on serum insulin-like growth factor and its binding proteins in athletes. *European Journal of Applied Physiology and Occupational Physiology*, **78**(6), 533-537.
- NINDL, B.C., KRAEMER, W.J., MARX, J.O., ARCIERO, P.J., DOHI, K., KELLOGG, M.D., & LOOMIS, G.A. (2001). Overnight responses of the circulating IGF-I system after acute, heavy-resistance exercise. *Journal of Applied Physiology*, **90**(4), 1319-1326.
- PAAVOLAINEN, L., HAKKINEN, K., HAMALAINEN, I., NUMMELA, A., & RUSKO, H.

(1999). Explosive-strength training improves 5-km running time by improving running economy and muscle power. *Scandinavian journal of medicine & science in sports*, **13**(4), 272-272.

PALMER, C.D., & SLEIVERT, G.G. (2001). Running economy is impaired following a single bout of resistance exercise. *Journal of Science & Medicine in Sport*, **4**(4), 447-459.

PRITZLAFF, C.J., WIDEMAN, L., WELTMAN, J.Y., ABBOTT, R.D., GUTGESELL, M.E., HARTMAN, M.L., VELDHUIS, J.D., & WELTMAN, A. (1999). Impact of acute exercise intensity on pulsatile growth hormone release in men. *Journal of applied physiology*, **87**(2), 498-504.

PURGE, P., JÜRIMÄE, J., & JÜRIMÄE, T. (2006). Hormonal and psychological adaptation in elite male rowers during prolonged training. *Journal of sports sciences*, **24**(10), 1075-1082.

ROECKER, K., MAYER, F., STRIEGEL, H., & DICKHUTH, H.H. (2000). Increase characteristics of the cumulated excess-CO<sub>2</sub> and the lactate concentration during exercise. *International Journal of Sports Medicine*, **21**(6), 419-423.

RAASTAD, T., BJORO, T., & HALLEN, J. (2000). Hormonal responses to high- and moderate intensity strength exercise. *European journal of applied physiology*, **82**, 121-128.

RUSH, J.W.E., WOODMAN, C.R., AAKER, A.P., SCHRAGE, W.G., & LAUGHLIN, M.H. (2000). Skeletal muscle blood flow and endurance exercise: limiting factors and dynamic responses, edited by R.J. SHEPHARD & P.-O. ÅSTRAND, 84-102. London: Blackwell Science Ltd.

ROHATAGI, S., BVE, A., MACKIE, A.E., & DERENDORF, H. (1996). Mathematical modeling of cortisol circadian rhythm and cortisol suppression. *European Journal of Pharmaceutical Sciences*, **4**, 341-350.

RYUSHI, T., HÄKKINEN, K., KAUKANEN, H., & KOMPI, A. (1988). Muscle fibre characteristics, muscle cross-sectional area and force production in strength athletes, physically active males and females. *Scandinavian Journal of Sports Sciences*, **10**, 7-15.

SAHLIN, K. (1986). Muscle fatigue and lactic acid accumulation. *Acta Physiologica Scandinavica*, **128** (suppl 556), 83-91.

SALE, D.G., MACDOUGALL, J.D., JACOBS, I., & GARNER, S. (1990). Interaction between concurrent strength and endurance training. *Journal of Applied Physiology*, **68**, 260-270.

SAUNDERS, U. A., PYNE, D. B., TELFORD, R. D., & HAWLEY, J. A. (2004). Factors affecting running economy in trained distance runners. *Sports Medicine*, **34**, 465-485.

SANCHEZ, J., PEQUIGNOT, J.M., PEYRIN, L., & MOND, H. (1980). Sex differences in the sympathoadrenal response to isometric exercise. *European Journal of Applied Physiology*, **45**, 147-154.

SIEGLER, J.C., BELL-WILLSON, J., MERMIER, C., FARIA, E., & ROBERGS, R.A. (2006). Active and passive recovery and acid-base kinetics following multiple bouts of intense exercise to exhaustion. *International journal of sports nutrition and exercise metabolism*, **16**, 92-107.

SCHUENKE, M.D., MIKAT, R.P., & MCBRIDE, J.M. (2002). Effect of an acute period of resistance exercise on excess post-exercise oxygen consumption: implications for body mass management. *European journal of applied physiology*, **86**(5), 411-417.

SCHWAB, R., JOHNSON, G.O., HOUSH, T.J., KINDER, J.E., & WEIR, J.P. (1993). Acute effects of different intensities of weight lifting on serum testosterone. *Medicine and Science in Sports and Exercise*, **25**, 1381-1385.

SMILLIOS; I., PILIANIIDIS, T., KARAMOUZIS, M. and TORKMAKIDIS, S. P. (2003). Hormonal responses after various resistance exercise protocols. *Medicine & science in sport & exercise*, **35**(4), 644-654.

SMITH, LL. (2000). Cytokine hypothesis of overtraining. A physiological adaptation to excessive stress? *Medicine and Science in Sports and Exercise*, **32**, 317-331.

SPENGLER, C.M., ROOS, M., LAUBE, S.M., & BOUTELLIER, U. (1999). Decreased exercise blood lactate concentrations after respiratory endurance training in humans. *European Journal of Applied Physiology & Occupational Physiology*, **79**(4), 299-305.

STOKES, K.A., NEVILL, M.E., HALL, G.M., & LAKOMY, H.K.A. (2002a). The time course of the human growth hormone response to a 6 s and a 30 s cycle ergometer sprint. *Journal of sports sciences*, **20**(6), 487-494.

TEGELMAN, R., JOHANSSON, C., HEMMINGSSON, P., EKLOF, R., CARLSTROM, K., & POUSETTE, A. (1990). Endogenous anabolic and catabolic steroid hormones in male and female athletes during off season. *International Journal of Sports Medicine*, **11**(2), 103-106.

TESCH. P.A., COLLIANDER, E.B., & KAISER, P. (1986). Muscle metabolism during intense, heavy –resistance exercise. *European Journal of Applied Physiology & Occupational Physiology*, **55**(4), 362-366.

TREMBLAY, M.S., & CHU, S.Y. (2000). Hormonal responses to exercise – methodological considerations. In Warren, M.P., & CONSTANTINI, N.W., *Sports endocrinology*. Humana Press Inc.: New Jersey.

TAIPALE, R.S., MIKKOLA, J., NUMMELA, A., VESTERINEN, V., CAPOSTAGNO, B., WALKER, S., GITONGA, D., KRAEMER, W.J., & Häkkinen, K. (2010). Strength training in endurance runners. *International Journal of Sports Medicine*, **31**, 468-476.

TREMBLAY, M.S., COPELAND, L., & VAN HELDER, W. (2005). Influence of exercise duration on post-exercise steroid hormone responses in trained subjects. *European Journal of Applied Physiology*, **94**, 505-513.

VANHELDER, W.P., RADOMSKI, M.W., & GOODE R.C. (1984). Growth hormone responses during intermittent weight lifting exercise in men. *European Journal of Applied Physiology and Occupational Physiology*, **53**, 31-34.

VIRU, A., SMIRNOVA T., KARELSON, K., SNEGOVSKAYA, V., & VIRU, M. (1996). Determinants and modulators of hormonal responses to exercises. *Biology of Sport*, **13**(3), 169-187.

VUORIMAA, (2007). Neuromuscular, hormonal and oxidative stress responses to endurance running exercises in well-trained runners. Jyväskylä: Publishing Unit, University Library of Jyväskylä.

WARREN, B.J., STONE, M.H., KEARNEY, J.T., FLECK, S.J., JOHNSON, R.L., WILSON, G.D., & KRAEMER, W.J. (1992). Performance measures, blood lactate, and plasma ammonia as

indicators of overwork in elite junior weightlifters. *International Journal of Sports Medicine*, **13**(5), 372-376.

WELTMAN, A., WELTMAN, J., SCHURRER, R., EVANS, W., VELDHUIS, J., & ROGOL, A. (1992). Endurance training amplifies the pulsatile release of growth hormone. Effects of training intensity. *Journal of applied physiology*, **72**(6), 2188-2196.

WELTMAN, A., WELTMAN, J.Y., ROY, C.P., WIDEMAN, L., PATRIE, J., EVANS, W.S., & VELDHUIS, J.D. (2006). Growth hormone response to graded exercise intensities is attenuated and the gender difference abolished in older adults. *Journal of applied physiology*, **100**(5), 1623-1629.

WIDEMAN, L., WELTMAN, J.Y., SHAH, N., STORY, S., VELDHUIS, J.D., & WELTMAN, A. (1999). Effects of gender on exercise-induced growth hormone release. *Journal of applied physiology*, **87**(3), 1154-1162.

WILLIAMS, K.R., & CAVANAGH, P.R. (1987). Relationship between distance running mechanics, running economy and performance. *Journal of Applied Physiology*, **63**, 1236-1245.