COMBINED STRENGTH AND ENDURANCE EXERCISE INDUCED FATIGUE AND RECOVERY

Eeva-Maria Kilpelänaho

Master's Thesis in Science of Sport

Coaching and Fitness testing

Spring 2012

Department of Biology of Physical Activity

University of Jyväskylä

Supervisor: Prof. Keijo Häkkinen

ABSTRACT

Kilpelänaho, Eeva-Maria. 2012. Combined strength and endurance exercise induced fatigue and recovery. Department of Biology of Physical Activity, University of Jyväskylä. Master's Thesis in Science of Sport Coaching and Fitness Testing, 81 pp.

Muscular fatigue is usually defined as the inability to maintain a given exercise intensity or adequate muscle force and therefore maximal isometric force, neural activation and fast force production capacity have often used determinants of acute fatigue. The purpose of the present study was to measure the acute effects of combined strength and endurance exercise on force production, muscle activation and work economy. In addition this study investigated the rate of recovery comparing the active and passive recovery models and the association of the initial fitness levels of subjects.

16 (27 ± 4 years old) male reservists performed combined strength (5x10x70%RM leg-press) and endurance (5x10 min marching) loading and following either active (5x10x30% leg press and 5x5min walking) or passive (seated) recovery. Bilateral maximal isometric force (MVC), force-time curve, muscle activation (EMG) and blood lactate were measured before and after the combined loading to determine the acute fatigue and recovery. In addition, work economy and heart rate were detected during the loading. The mean (± SD) magnitude of combined exercise induced loss in MVC was 14.2 ± 9.5 % (n=16) (p=0.001), and the force-time curve (n=16) shifted significantly (p<0.05-0.01) to the lower level. Significant decreases (p< 0.05 - p < 0.01) occured also in the EMG values, and the mean work economy (VO_2/km/h) decreased by 11 ± 11 % (p=0.001) during the loading. The mean blood lactate increased (p=0.001) to 4.02 ± 1.89 mmol/l but recovered during the following hour after the test. MVC, EMG and HR recovered totally in both groups by the next morning. In addition, a significant (p<0.05) association between the VO_{2max} of the subjects and the magnitude of force decrease was detected. The VO_{2max} of the subjects also showed an association with the heart rate recovery.

The present findings suggest that strenuous combined strength and endurance loading results in acute fatigue in the neuromuscular system leading not only to the decreased force production capacity of the muscles but also to a decrease in the muscle activation of the exercised muscles. The study also showed that the better the aerobic fitness level of the subjects the smaller was the neuromuscular fatigue and faster the physiological recovery rate. In addition, the results of the present study suggest that the active recovery might have beneficial effects on faster recovery of neuromuscular performance. Studying the acute performance decrements and recovery by simulating the physical demands of combined strength and endurance exercise may provide some valuable insight into the development of training programs for military and sport missions and for optimizing soldiers and athletes' capacity for physical performance.

Keywords: combined exercise, force decrement, electromyography, work economy, blood lactate, active recovery, passive recovery

CONTENTS

1 INTRODUCTION

Number of sport activities and tasks and missions carried out by military personnel require often efforts of combination of strength and endurance exercise (de Souza et al. 2007, Nindl et al. 2002). In military operations soldiers are also often exposed to various stressors, such as prolonged and strenuous physical exercise, energy and fluid deficiency, and sleep deprivation (Bulbulian et al. 1996, Nindl et al. 2002, Kyröläinen et al. 2008). Researchers have been studying the relationship between physical exercise and fatigue for more than a century and currently it is known that exhaustive exercise can cause fatigue, which causes acute performance decrements but it is a very complex phenomenon (Brooks & Fahey 1984, 710, Fitts 2008, Ament &Verkerke 2009). In addition, there are several controversies in the literature about the physical performance during the combined strength and endurance exercise (Brunetti et al. 2008).

Previous studies have examined the effects of exhaustive physical efforts on the performance of military personnel over longer periods of time (Nindl et al. 2002, Kyröläinen et al. 2008) but the acute performance decrements due to combined exercise tasks have not yet been examined. More developed picture of neuromuscular, hormonal, and metabolic mechanisms behind fatigue is needed (Ament &Verkerke 2009). Furthermore, another critical question is how individuals would be able to avoid overstress and accelerate recovery as effectively as possible, as previous studies have shown that complete recovery of neuromuscular system can take even days after the exhaustive loading (Häkkinen & Komi 1986, Häkkinen 1993, Häkkinen 1994).

Therefore, the purpose of the study is to determine the acute combined strength and endurance exercise induced fatigue and to compare active and passive recovery models to the recovery rate in reservist men. In addition, possible relationship between individual fitness level of the subject and the rate of fatigue and recovery will be examined.

2 SPECIFICITY OF COMBINED STRENGTH AND ENDURANCE EXERCISE

The combination of strength and endurance exercises in the same session is called concurrent training (Leveritt et al. 1999) and has been used in an attempt to improve performance in particular sports (Mikkola et al. 2007), military tasks (Kraemer et al. 1995) and with elderly people (Ferketich et al. 1998, Holviala et al. 2010) (figure 1).

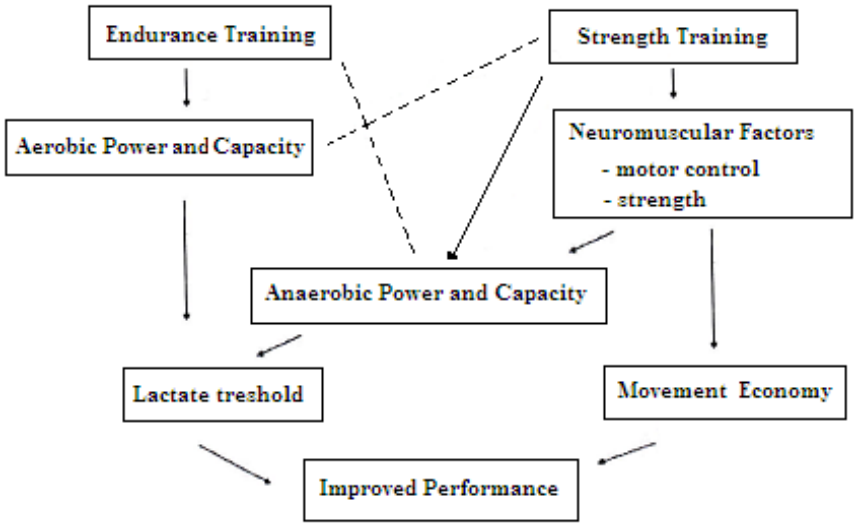

FIGURE 1. Combined strength and endurance exercise can have potential interrelated mechanisms for developing for example endurance performance or military skill tasks (modified from Paavolainen et al. 1999).

However, multiple studies have come to the conclusion that concurrent strength and endurance training results in several adaptations that are different from either strength or endurance training alone because of their divergent nature of physiological stimuli directed to skeletal muscle (Hickson 1980, Kraemer et al. 1995, Bell et al. 2000, Häkkinen et al. 2003). Due to the specific physiological, biochemical, and molecular mechanisms of combined exercise (Nader 2006), it has been shown that combined training may lead to lower strength gains (Hickson 1980, Bell et al. 2000, Doherty & Sporer 2000, Häkkinen et al. 2003) as well as to lower magnitude of endurance

development (Kraemer et al. 1995, Leveritt et al. 1999) compared with either exercise mode alone (Izquierdo et al. 2005, Hawley 2009, Mikkola et al. 2007, Chilibeck et al. 2002) (figure 2).

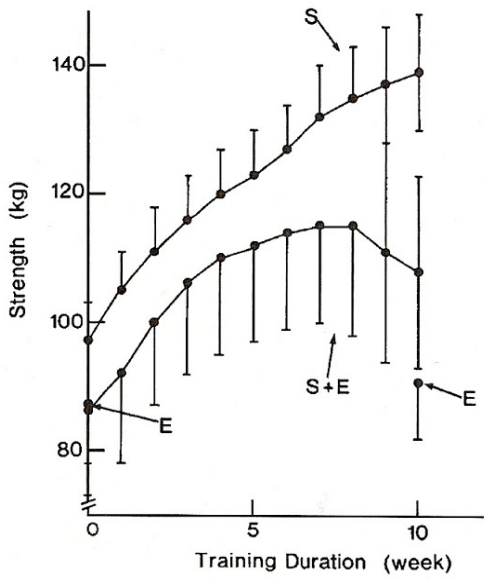

FIGURE 2. Interference of strength development by concurrent strength and endurance training compared to strength training mode alone (Hickson 1980).

However, some studies indicate that the interference effect may be true only when the training is prolonged or overall frequency and/or volume of training is high (Izquierdo et al. 2005, Häkkinen et al. 2003). Because the interference effect is always influenced by volume-, intensity- and duration of the training program as well as the training background and the age of the subjects (Chtara et al. 2005), these different results of concurrent training and its chronic effects seem to be related to the different experimental protocols that have been used (Doherty & Sporer 2000, Bell et al. 2000). In addition, though lots of studies have examined the concurrent training and its chronic effects, little is known about the focus of this study; the acute responses that occur during or immediately after combined exercise (Brunetti et al. 2008). Regarding combined exercise induced acute fatigue, the most important finding of previous studies appear to be hypothesis about the influences of intrasession order to combined exercise induced adaptations. According to that hypothesis the first activity induced fatigue would reduce or change the physiological adaptations to the second activity. (Doherty & Sporer 2000, Chtara et al. 2005.)

3 ACUTE FATIGUE RESPONSES TO EXHAUSTIVE EXERCISE

3.1 The overall mechanism of neuromuscular fatigue

Intensive continuous or intermittent muscular work will lead to a momentary decrease in the performance capacity of the neuromuscular system (Häkkinen & Komi 1986, Häkkinen et al. 1988a) which can be originated to the central or peripheral side of nervous system (Bigland-Ritchie & Woods 1984, Fitts 2008). Figure 3 presents the possible sites of muscular fatigue. Generally central fatigue alters the capacity of the central nervous system to drive the motor neurons, whereas peripheral fatigue occurs within the muscle. (Yeung et al. 1999, Kent-Braum 1999, Sidhu et al. 2009).

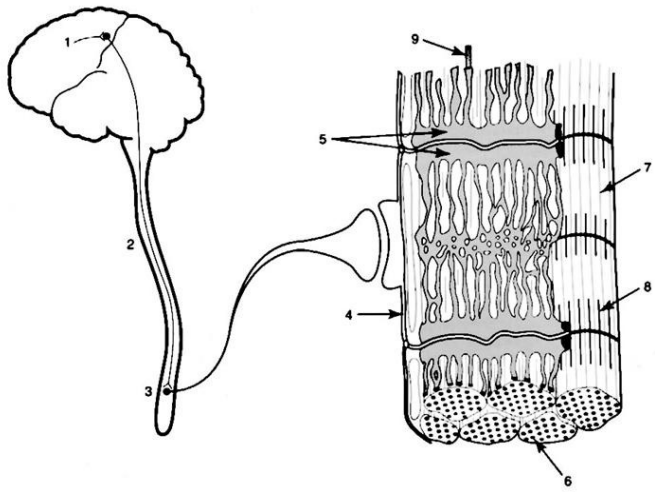

FIGURE 3. Central sites of muscular fatigue include: 1) excitatory input to motor cortex, 2) excitatory drive to lower motor neuron, 3) motor neuron excitability, 4) neuromuscular transmission. Peripheral sites within the muscle contain 5) sarcolemma excitability, 6) excitation contraction coupling, 7) contractile mechanism, 8) metabolic energy supply (modified from Enoka 2002, 374).

In short term exercise the fatigue is mainly caused by peripheral factors like metabolic or muscular damage (Denadai et al. 2007), whereas it has been suggested that during prolonged exercise muscle fatigue is generated by both central and peripheral factors (Millet & Lepers 2004, Nummela et al. 2008a). Still the identification of a fatigue cause is not a simple matter, as it is often difficult to separate causality from concurrent appearance (Brooks & Fahey 1984, 710). For example, if intracellular metabolites cause a failure of skeletal muscle contractile function during exhausting exercise, how would the fact that subjects are able to increase their running pace near the end of an exercise bout be explained (Sharwood et al. 2003)? Previous studies have, however, shown how brain and the central nervous system regulate the extent of output drive by increasing or decreasing voluntary motor neuron activation. Numerous studies have shown how peripheral factors such as increasing plasma acidosis (Cairns 2006), tissue damage and pain (Sharwood et al.2003), elevations in brain, core and local tissue temperatures (Ament &Verkerke 2009, Duffield et al. 2009) or perception of the exercise (Calbet et al. 2009) reduce central nervous system drive to active musculature, which has seen as a preventative mechanism. Similarly decreases in cerebral oxygenation or brain catecholamines may introduce the sensations of fatigue and the sense of effort during exercise and contribute to central fatigue (Sidhu et al. 2009).

3.2 Decrease in force production and muscle activation

Muscle fatigue can be defined as an inability to maintain the level of force production or as a reduction in the maximum force that a muscle can exert (Komi & Tesch 1979, Bigland-Ritchie & Woods 1984). Failure of force production may occur at the various sites along the pathway from the central nervous system to the intramuscular contractile machinery (figure 3) (Kent-Braum 1999, Millet & Lepers 2004) and is differing according to the types of contraction involved, the muscular groups tested, the training background of the individual and the overall volume and intensity of exercise (Häkkinen 1993, Linnamo et al. 2000, Skof & Strojnik 2005, Nummela et al. 2008a). In combined exercise both intensive muscular work, such as hypertrophic or heavy resistance loading (Ahtiainen et al. 2003, Izquierdo et al. 2009), and long-duration

exercise as running (Sharwood et al. 2003, Skof & Strojnik 2005) lead to fatigue which include decreased maximal force, neural activation and velocity of force production.

3.2.1 Reduction in maximal voluntary muscle force

Exercise-induced muscle fatigue is traditionally measured by isometric maximal voluntary contraction (MVC) (Byrne & Eston 2002, Izquierdo et al. 2009). Previous studies (Häkkinen 1993, Häkkinen 1994, Häkkinen & Pakarinen 1995, Ahtiainen et al. 2003) have distinctly shown how isometric strength is reduced after a heavy resistance and hypertrophic exercises. For example in the study of Häkkinen (1994), strenuous resistance loading (squatting 10*10*70 % of 1 RM) led to approximately 47% decrease in isometric MVC. The study of Tesch & Wright (1983) showed an average of 67 % decrease in voluntary muscle strength after 50 consecutive performed maximal voluntary knee extensions. Figure 4 in turn represents (24,1 ±14,4%) the decrease of male athletes' maximal isometric force after heavy resistance (20*1*100% of 1RM) loading (Häkkinen 1993).

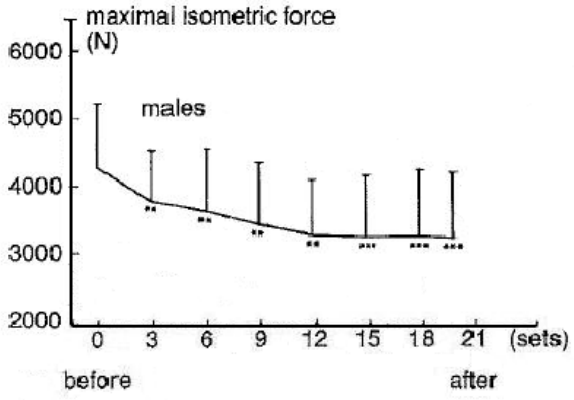

FIGURE 4. The gradual decrease of male athletes maximal isometric leg extension force as recorded immediately after the fatiguing loading (modified from Häkkinen 1993).

Dealing with combined strength and endurance exercise, the order effect can also have an additional impact on the fatigue response. For example, both the studies of Leveritt & Abernethy (1999) and Sporer & Wenger (2003) verified the negative influence in the strength production of lower limbs after an intermittent or continuous activity performed in the cycle ergometer. Also the study of de Souza et al. (2007) noticed the reduced strength endurance performance (maximum repetitions at 80% of 1RM) after 5-kilometer run when aerobic exercise was performed intermittently (1:1 minute at VO_{2max}). That decrease in the production of strength has been attributed to many factors, as the lack of time for the muscles to recover from the previous exercise (Chtara et al. 2005) and the decrease in neural activation (Brunetti et al. 2008).

Another possible explanation is that the endurance exercise involving relatively large amounts of eccentric muscle activity may impair performance in a subsequent strength exercise session (Leveritt et al. 1999, Bell et al. 2000). A number of studies have shown how an aerobic exercise with large eccentric component (i.e. running) has a tendency to promote greater and longer lasting strength loss than activities with predominantly concentric actions which is probably due to muscle damage (Byrne & Eston 2002, Denadai et al 2007). In human running considerable impact loads occur when contact is made with the ground (figure 5).

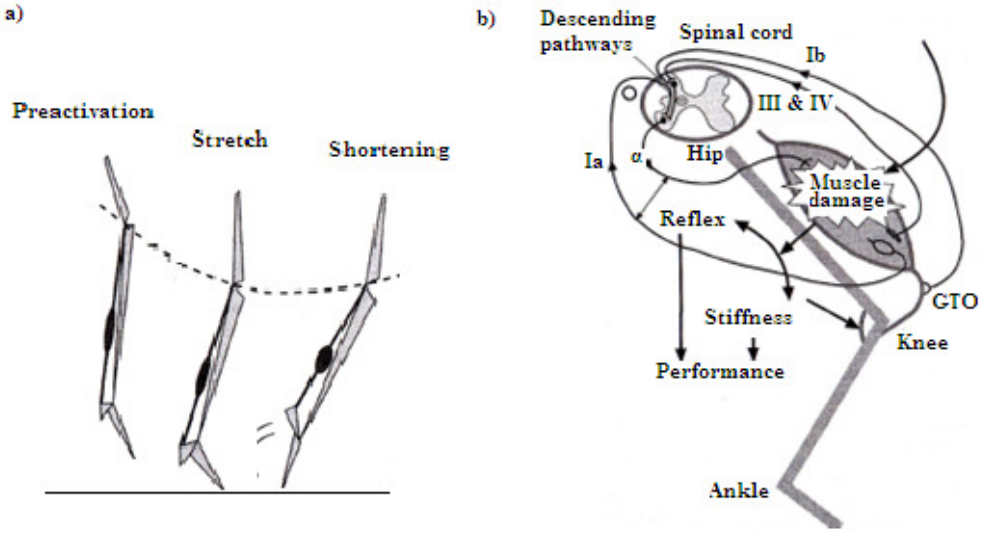

FIGURE 5. In stretch-shortening cycle exercise induced muscle damage the stretch-reflex sensitivity decreases, muscle (and joint) stiffness regulation becomes disturbed and the performance decreases. (Figure a) modified from Komi & Nicol 2000 and b) Horita et al. 1999.)

For example, both Sharwood et al. (2003) and Nummela et al. (2008a) have found linear decreases in force production (MVC) and iEMG due to 5 km running performances. Figure 5 represents the coupling between stretch-shortening cycle exercise-induced muscle damage and performance decrements (Nicol et al. 1996, Horita et al. 1999, Komi & Nicol 2000, Millet & Lepers 2004).

3.2.2. Changes in electromyographical activity

Fatigue-related decreases in muscle voluntary activation to maintain a given muscle power output (i.e. dynamic task failure) have been exclusively assessed by the measurement of the EMG signal during maximal voluntary isometric contractions (Viitasalo & Komi 1977, Komi & Tesch 1979, Izquierdo et al. 2009). Usually fatigue leads to changes in the size and height of the EMG amplitude (due to exercise induced decrease of relaxation velocity) and to increase in the interval between the onset of muscle electromyographic activity and developed force (EMD) (figure 6) (Yeung et al. 1999, Komi & Nicol 2000).

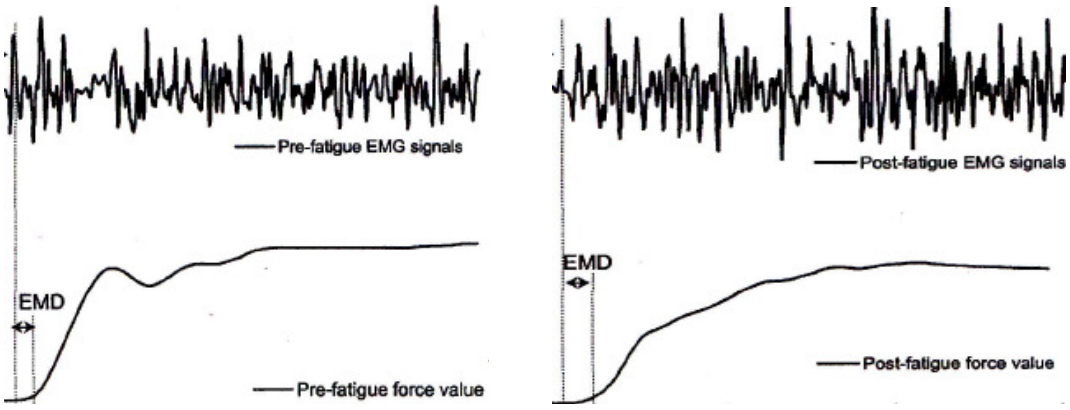

FIGURE 6. Force production, EMG and the electromechanical delay (EMD) measured by isometric maximal voluntary contraction (MVC) before and after the fatiguing contraction. (Yeung et al. 1999.)

Also previous studies reporting decreases in strength following heavy resistance and hypertrophic loadings are likely related to muscle activation and the recruitment of muscle fibres (Häkkinen 1993, Häkkinen 1994, Linnamo et al. 2000, Ahtiainen et al. 2003). For example, both squatting ten times ten repetitions about 70 % of 1 RM (Häkkinen 1994) and performing one repetition maximum 20 times in another study of Häkkinen (1993) led to significant decrease in knee extensors maximal iEMG. When Häkkinen (1992) studied acute neuromuscular responses to high but submaximal loading (two strength trainings performed the same day), only slight changes took place in the maximal neural activation (iEMG) of the exercised muscles.

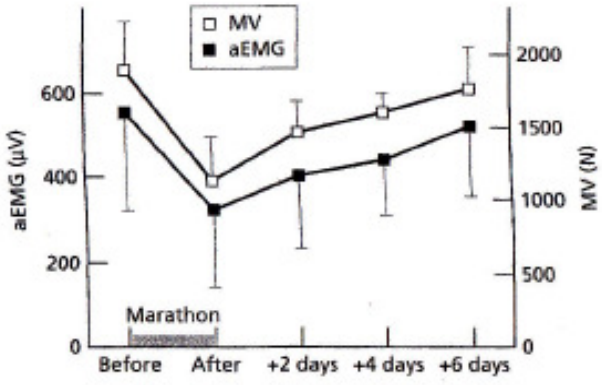

FIGURE 7. Marathon running caused dramatic reduction and long lasting impairment of maximum EMG and force of the isometric knee extension (Pullinen et al. 1997).

Although the effects of fatigue on EMG activity in submaximal exercises are not as clear as the reduced EMG activity in maximal efforts, a decrease in EMG activity for lower limb muscles also after prolonged runs has been recorded (figure 7) (Paavolainen et al. 1997, Sharwood et al. 2003, Millet & Lepers 2004, Nummela et al. 2008a). Also the studies regarding combined exercise have discussed the possibility that neural factors and motor unit recruitment may have a significant role in restricting strength development in concurrent training (Brunetti et al. 2008). In the study of de Souza et al. (2007), it has been suggested that an explanation for combined exercise induced acute interference effect was the motor unit pool activation during exercises. If strength exercise and endurance exercise could activate the same motor unit pool, acute interference effect was maximized (de Souza et al. 2007). Also in the study of Häkkinen et al. (2003) effects of concurrent training on rates of force development were assumed

to have been a consequence of neural and muscle components, limiting rapid voluntary neural activations.

3.2.3 Decrease in rate of force development

During exercise-induced acute fatigue both the integrated electromyogram (EMG) and the mean firing rate of individual motor units decline progressively with declining maximum force generation (Bigland-Ritchie & Woods 1984). Those changes in the velocity of force production can be indicated by the right and the downward shift in the force-time curve (figure 8) (Häkkinen 1990, Häkkinen & Komi 1986, Häkkinen et al. 1988a, Häkkinen 1992, Häkkinen 1993).

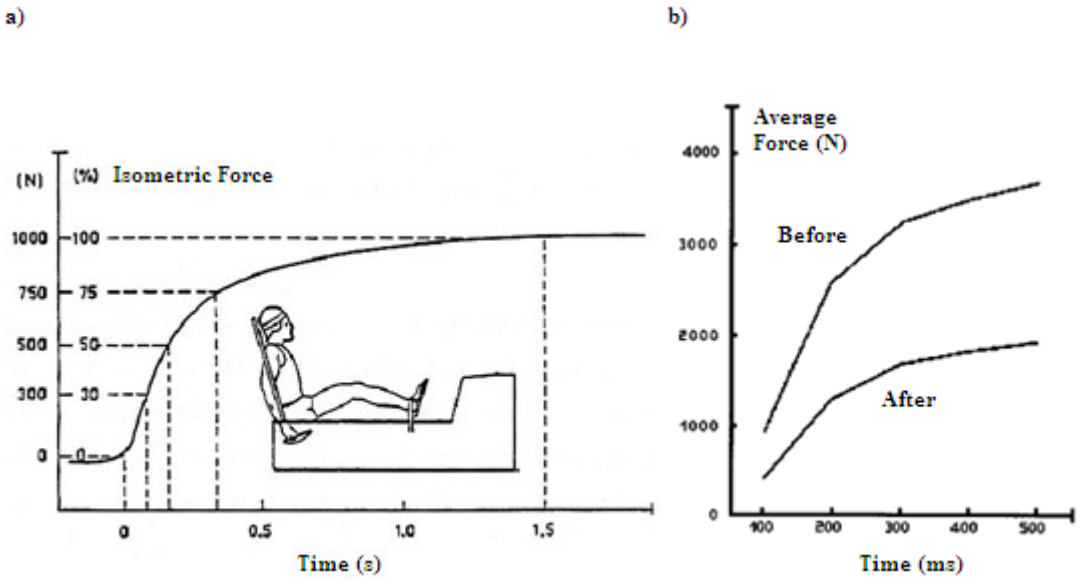

Figure 8. a) Force-time curve representing the rate of force development (Häkkinen 1990) and b) the mean changes of velocity of force production due to fatiguing (squatting 10*10*70% of 1RM) loading of male strength athletes (Häkkinen 1994).

Linnamo (1993, 53) has found 27 % decrease in maximal rate of force development and Häkkinen (1994) 49.3±11.5 % decrease in force-time curve after the fatiguing knee extension involved loadings. In addition, an emphasized downward shift in the force-

time curve (Komi & Viitasalo 1977) and vertical jump performance (Paavolainen et al. 1994, Byrne & Eston 2002, Kubo et al. 2005) has been found after eccentric actions involved exercises due to changed muscle tension and neural activation (Byrne et al. 2001). Also combined strength and endurance exercise have been found to cause significant decrease in rapid force production regardless of the order of exercises in the study of Schumann (2011, 48).

3.3 Changes in intracellular environment

Peripheral factors within the muscles have often been associated with the failure to maintain a given level of muscle force under conditions of exercise-induced fatigue (Mainwood & Renaud 1985, Sahlin et al. 1978). Fatigue during muscular exercise can be due to specific depletion of key metabolites in muscle or to the accumulation of other metabolites, which can affect the intracellular environment (Brooks & Fahey 1984, 701). Typically accumulation of lactate, H^+ inorganic phosphate (Pi) and ammonia within the active muscles have been seen leading to metabolic inhibition of the contractile process and the failure in excitation-contraction coupling (Kent-Braum 1999, Izquierdo et al. 2009). Additionally, glycogen depletion has been shown to reduce following performance (Sahlin et al. 1999, Leveritt et al. 1999, de Souza et al. 2007).

3.3.1 Increased metabolite accumulation

When part of the energy requirement is produced by glycolysis causing metabolic end products, energy production and force generation are thought to be impaired (Christmass et al. 1999). Moreover, exercise characteristics may influence production of metabolites (de Souza et al. 2007). For example, previous studies using hypertrophic and heavy resistance loadings have shown significant increases in lactate concentration (Linnamo et al. 2000, Ahtiainen et al. 2003). In the study of Linnamo et al. (2000), leg

press exercise (5*10*70%) elevated lactate concentration to 4.95±0.81 mmol/l, whereas in the study of Häkkinen (1994) lactate raised to even 15.0 ± 4.0 mmol/l after the fifth set and stayed there until the end of the loading (10*10*70%). Additionally, Ahtiainen et al. (2003) suggested the longer the concentric phase of the dynamic muscle actions the higher the load used, the more the blood circulation and accumulated metabolic waste products more in the muscle cells decreased. Also prolonged sufficient intensive aerobic activities have increased lactate concentrations (Viru et al. 2001). In the studies of Skof & Strojnik (2005) and Denadai et al. (2007), intensive running at anaerobic threshold raised blood lactate concentrations to the levels of 5.9 mmol/l and 5.0 mmol/l.

Also in combined exercise elevated blood lactate levels are evident after intensive muscular work (Leveritt et al. 1999, Leveritt et al. 2000, Brunetti et al. 2008, Holviala et al. 2010). De Souza et al. (2007) have concluded that accumulation of metabolites can partly explain the acute interference phenomenon during combined exercise. In the study of Leveritt & Abernethy (1999), plasma lactate concentrations were significantly higher (6.16 ± 2.28 mmol/l) at the beginning of strength exercise - 30 min after high-intensity endurance bout - when compared to a condition without endurance bout (0.50 ± 0.45 mmol/l). Schumann (2011, 49) has found significantly increased blood lactate levels during both endurance running and strength loading in combined exercise, and there were no order effect recognized in males. Similarly, in the study of Brunetti et al (2008) blood lactate concentrations increased significantly during the both endurance and strength exercises despite the performing order.

Generally, the lactate accumulation is associated with a rise in muscle and blood H^+, which has been related directly or indirectly to local fatigue and decline in pH (Baldari et al. 2007). Low pH have been seen to impair force production by reducing the number of cross bridges and the force per cross bridge (Cairns 2006, Fitts 2008). In the pioneering work of Mainwood & Renaud (1985) in figure 9, isolated frog muscle was stimulated to fatigue which resulted in intracellular proton load and at the same time maximum isometric force suppressed by 70-80%. An additional change through decreasing pH is reduction in a conduction velocity/surface EMG of skeletal muscle

caused by inhibition of the release of Ca^{2+} into the sarcoplasmic reticulum and reduced Ca^{2+} affinity for troponin (Komi & Tesch 1979, Mannion & Dolan 1996).

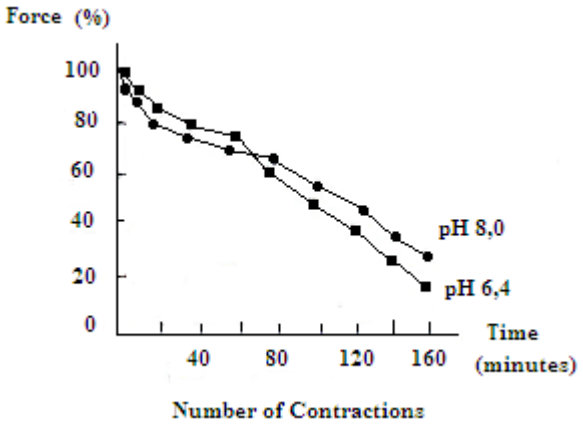

FIGURE 9. Reduction in force output due to decrease in extracellular pH (modified from Mainwood & Renaud 1985).

3.3.2 ATP and glycogen depletion

Failure to maintain strength level under exercise-induced fatigue has also been associated with exercise induced decline in muscle adenine nucleotide stores, mainly through a reduction in muscle ATP content (Sahlin et al. 1999, Izquierdo et al. 2009). For example, hypertrophic type resistance exercise depletes creatinephosphate (CP) stores almost completely and reduces ATP stores 30-40 % from pre-exercise value (figure10) (Tesch et al. 1986). According to Komi & Tesch (1979), an adequate availability of ATP and the accumulation of intracellular ADP in the myofibrillar regions, is responsible for impaired contractile function decreasing the speed of cross-bridge interaction of the muscle cell. Increased ADP also creates a microenvironment (i.e. high ADP/ATP ratio) that is unfavorable for ATP-ase function, and as a consequence, sarcoplasmic reticulum Ca^{2+} pump function may be diminished (Chilibeck et al. 2002, Cooke et al. 2009). The decreased rate of Ca^{2+} transport back into the sarcoplasmic reticulum in turn decreases the speed of force relaxation at the end of a contraction (Piitulainen et al. 2008).

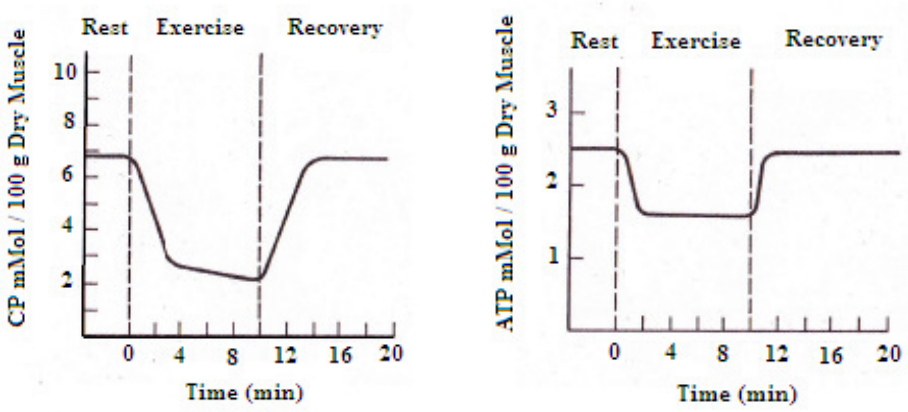

FIGURE 10. Since exercise starts adenosine triphosphate (ATP) level declines but is well maintained (largely at the expense of CP) until the fatigue point, when ATP and CP become both depleted (modified from Bergström 1967 in Brooks & Fahey 1984, 705.)

Plenty of studies have shown that catabolism of total adenosine nucleotides is also related to glycogen depletion, when successive bouts of either strength or endurance exercise may produce chronically low muscle-glycogen levels, which could retard or impair subsequent performances (Sahlin et al. 1999, Leveritt et al. 1999, Nader 2006, Osborne & Scheider 2006). Therefore, Brunetti et al. (2008) and de Souza et al. (2007) suspected that if the intensity of an aerobic exercise is high enough to deplete muscle glycogen content, the strength performance will be greatly affected in combined exercise. Also in the study of Leveritt & Abernethy (1999), a programme of carbohydrate restriction and muscle glycogen depletion actually showed a reduction in isometric strength performance.

3.4 Changes in serum hormone concentration

A human body responses to intense muscular work also through its endocrinal function (Kuoppasalmi et al. 1980, Adlercreutz et al. 1986). Heavy resistance exercise is known to be a potent stimulus for acute increases in the concentrations of circulating hormones and to increase in serum testosterone and growth hormone concentrations (Linnamo et

al. 2005, Häkkinen & Pakarinen 1993, Ahtiainen et al. 2003, Ahtiainen et al 2005, Kraemer & Ratamess 2005) but the acute responses to endurance exercise seem to vary across individuals (Viru et al. 2001). It has been established that fatigue from long endurance activity may induce a "resetting" of the pituitary-adrenocortical component of the endocrine system, which can be expressed by either intensified or suppressed endocrine functions (Viru et al. 2001, Ahtiainen et al. 2009).

Anabolic and catabolic hormones (i.e. testosterone and cortisol) may play a vital role also in responses to simultaneous strength and endurance exercise (Kraemer et al. 1995, Bell et al. 2000). It have been demonstrated that combined exercise produce differential hormonal responses compared with strength and endurance exercises alone (Kraemer et al. 1995, Chtara et al. 2005). High-intensity strength exercise results in a potent stimulus for muscle cell hypertrophy that appears mediated via increases in protein synthesis and accretion of contractile proteins (Ahtiainen et al. 2003, Linnamo et al. 2005). Conversely, an oxidative endurance exercise stress causes muscle to respond in an opposite fashion by ultimately degrading and sloughing myofibrillar protein to optimize oxygen uptake kinetics (Kuoppasalmi et al. 1980). Therefore, Nader (2006) has proposed that acute concurrent strength and endurance exercise does not promote optimal activation of pathways that simultaneously promote both anabolic and endurance responses. This finding can be partly explained by fatigue after the first activity which may influence the endocrinal adaptations to the second activity (Viru et al. 2001, Chtara et al. 2005).

The results of Linnamo et al. (2005) suggested that increases in serum growth hormone appear to be greatest during hypertrophic types of exercises when using rather high numbers of repetitions and sets when in the case of serum testosterone, the higher loads cause more dramatic increase in hormone level compared to moderate loads. Figure 11 presents two hypertrophic type resistance exercise loadings induced increases of serum testosterone, free testosterone and cortisol levels in strength athletes. According to Ahtiainen et al. (2003), these acute increases in serum testosterone concentrations may be caused by the influence of the increased circulation in the testicles, activation of the

sympathetic nervous system, increased lactate accumulation and/or luteinizing hormone concentrations.

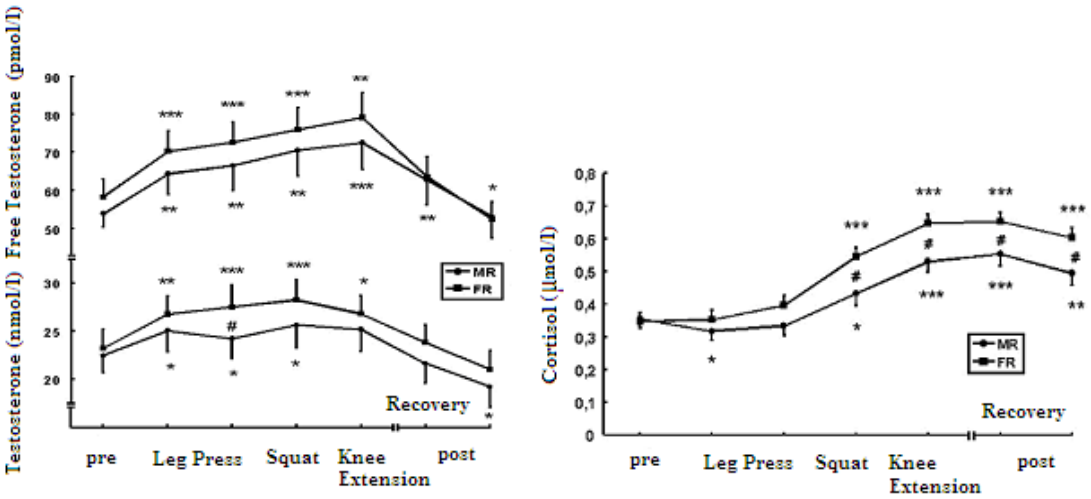

FIGURE 11. Maximum repetition (MR) and forced repetitions (FR) loadings induced changes in serum testosterone, free testosterone and cortisol concentrations after 4 sets of leg presses, 2 sets of squats and 2 sets of knee extensions (with 12 RM in MR group) (Ahtiainen et al. 2003).

Exercise-induced cortisol response in turn may be due to glycolytic demands of the exercise, stimulated effect of catecholamines and/or a consequence of neural control of muscle work (Ahtiainen et al. 2003). According to the study of Adlercreutz et al. (1986), intense long term physical exercise led to an increase in plasma cortisol and a decrease in plasma testosterone concentration. Also Kuoppasalmi et al. (1980) and Tremblay et al. (2005) found that long-term running caused considerable increases in mean plasma cortisol and androstenedione. Similarly, Bell et al. (2000) have concluded that concurrent strength and endurance training led to an elevated catabolic state in women compared to performing the same strength or endurance training separately or in comparison to men. Elevated catabolic state was indicated in their study by higher concentrations of cortisol combined with no change in the concentration of anabolic hormones such as testosterone or growth hormone (Bell et al. 2000). In the study of Viru et al. (2001), a two-hour run caused increases of growth hormone and variable responses in the concentrations of cortisol and testosterone (figure 12).

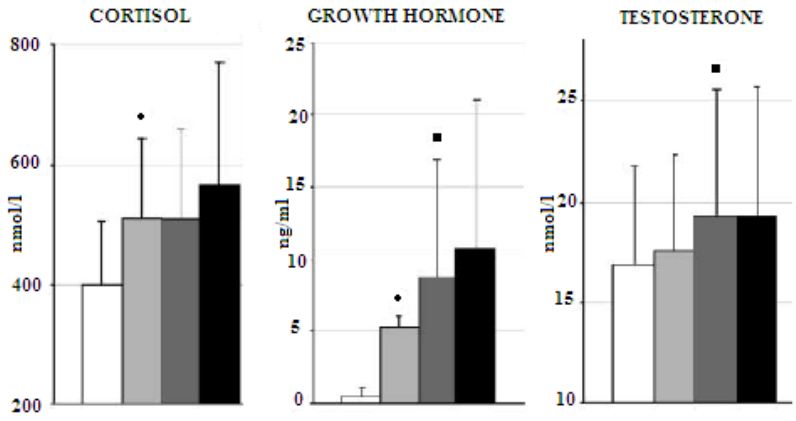

FIGURE 12. The pattern of the increases (from white pre-exercise value) in cortisol, growth hormone and testosterone as influenced by the two 10 minutes exercise tests (light grey and black) and two-hour run (dark grey) (modified from Viru et al. 2001).

Nevertheless, the acute endocrinal responses are always highly dependent on many factors including subject's age, rest intervals between sets, the total amount of work, and the size of the muscle mass activated (Linnamo et al. 2005, Häkkinen & Pakarinen 1993, Ahtiainen et al. 2003, Ahtiainen et al. 2005, Senna et al. 2008). Acute hormonal responses may also be related to metabolic changes during heavy resistance exercise. Linnamo et al. (2005) have shown that hormonal changes were related to the lactate response caused by the exercise, and inversely blood lactate tend to increase more when the number of repetitions were high with high loads than when loads or the number of repetitions were lower.

4 RECOVERY FROM EXHAUSTIVE EXERCISE

4.1 Recovery of neuromuscular performance

The recovery from acute neuromuscular fatigue depends on the magnitude of the acute-fatigue induced decrease in performance and the specific type of fatiguing load (Tesch & Wright 1983, Häkkinen et al. 1988a, Häkkinen 1993). In addition post-exercise related recovery is dependent on the extra- and intramuscular properties (Denadai et al 2007, Sidhu et al. 2009). Therefore, recovery can be divided into acute and prolonged (Siegler et al. 2006). The acute recovery happens first 30 seconds immediately after exercise and is considered to be a partial return of force production which is connected to restoring of ATP and CP. The next, so-called slow phase of recovery includes following 10-50 minutes and is related to removal of lactate. (Fitts & Holloszy 1976.)

4.1.1 Recovery of force production

Previous studies have shown how isometric strength has reduced after intensive muscular work and recovery takes from hours to several days (Häkkinen 1993, Häkkinen1994, Häkkinen 1995, Linnamo et al. 2000). Expressing acute recovery, Tesch & Wright (1983) registered 37 % recovery in voluntary muscle strength during very brief 40 – s recovery period after 50 consecutive performed maximal voluntary knee extensions. Several studies have shown that in prolonged recovery phase the maximal force recover fast but not completely during the first hour of rest (Häkkinen 1993, Linnamo 1993, 68, Häkkinen 1994, Häkkinen 1995). Figure 13 shows how isometric maximal force recovered significantly during the first hour, stayed impaired during the next day and recovered completely two days after the hypertrophic type loading (Häkkinen 1994). In turn, in the study of Ahtiainen et al. (2003) maximal isometric force stayed significantly lowered even 3 days after the heavy resistance exercise.

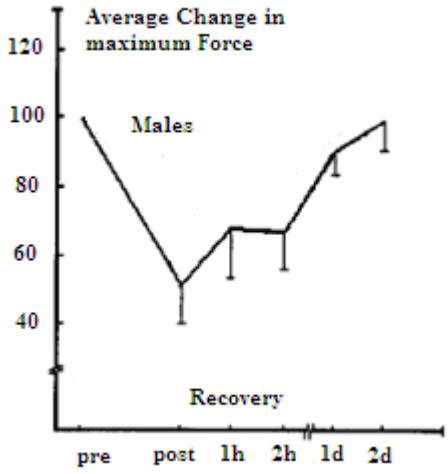

FIGURE 13. Recovery of bilateral maximal isometric force immediately, 1 hour (1h) 2 hours (2h) 1 day (1d) and 2 days (2d) after 10*10*70% of 1RM resistance exercise session (modified from Häkkinen 1994).

Millet & Lepers (2004) suggested that the strength loss after prolonged exercise is related to the exercise duration. In the study of Skof & Strojnik (2005) maximum isometric knee extension (MVC) remained decreased over a two-hour recovery period after intensive aerobic running (6-kilometer at anaerobic threshold). Instead, marathon running (Pullinen et al. 1997) and fatiguing stretch-shortening cycle involved exercises have caused impaired force production capacity for several days (Nicol et al 1996, Horita et al. 1999, Avela et al. 2001, Komi & Nicol 2000, Dousset et al. 2007). Studying the combined exercise, Sporer & Wenger (2003) found a significant reduction in strength endurance (maximum number of repetitions at 75% of 1RM) until 8 h after aerobic activity and the complete recovery happened after 24h of rest. Also Leveritt & Abernethy (1999) found the negative influence in the force production of squat exercise, after a cycle ergometer activity. The study of Leveritt et al. (2000) in turn, did not identify significant decrease in the force production after 50-minute exercise in a cycle ergometer, but resistance exercise was conducted as late as 8h and 32h after an aerobic activity which might be long enough time to achieve full recovery from aerobic exercise.

The results from the recovery of fast force production are in turn controversial. For example in the study of Linnamo (1993, 70), the velocity of force production was recovered during the first hour after hypertrophic type loading but an hour later it was, however, significantly reduced compared to pre-exercise value. Also Byrne & Eston (2002) found nonlinear manner of recovery of vertical jump performance after squatting exercise (10*10*70% of body mass load), when significant three-day reductions in vertical jump performances were immediate and long-lasting, possibly being explained by the observed reduction in strength of the knee extensors. Also endurance exercises have caused decreased muscle power (Viru et al. 2001). For example, in the study of Vuorimaa et al. (1999) vertical jump height (CMJ) reduced 3 days after a 28-minute intermittent middle-distance running.

4.1.2 Recovery of neural activation

Earlier studies have found several changes in EMG during neuromuscular fatigue and recovery (Byrne et al. 2001, Millet & Lepers 2004). The study of Ahtiainen et al. (2003) showed controversial results of neuromuscular recovery as the decreased maximal iEMG recovered during the first 24h of rest but decreased thereafter (figure 14).

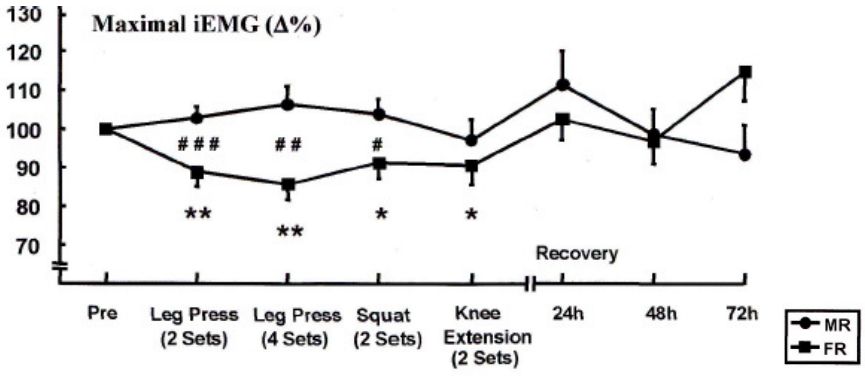

FIGURE 14. The recovery of iEMG after the hypertrophic type maximum repetitions and forced repetitions loadings (modified from Ahtiainen et al. 2003).

Also in the study of Linnamo (1993, 74-76), iEMG started to recover one hour after the hypertrophic loading but was not completely recovered even two days later. Interestingly, Behm & St-Pierre (1997) found duration induced impairments in muscle activation. Increased time to fatigue resulted in greater decreases in muscle activation (Behm & St-Pierre 1997). For example, in the study of Skof & Strojnik (2005) a 6-kilometer run caused an impairment of two hours in neural activation, whereas, in the study of Pullinen et al. (1997) a 42-kilometer marathon run caused six days impairment in EMG. According to Millet & Lepers (2004) spinal adaptation, such as inhibition from type III and IV group afferents or disfacilitation from muscle spindles (figure 5), contributes to the reduced neural drive after the prolonged exercise. Neural activation deficit is also one suggested explanation concerning combined exercise. In order to produce a maximum strength, all available motor units should be recruited, but they might be fatigued by the previous exercise (de Souza et al. 2007). For example, the study of Schumann (2011, 48) showed how the rapid force production within 500ms during MVC was decreased after combined strength and endurance exercise and there was no order effect recognized.

4.2 Recovery of energy stores and pH

Knowing that the strength performance is highly dependent on the anaerobic energetic metabolism, following priorities of the recovering muscles is to restore phosphocreatine and ATP stores and acid-base balance (Bogdanis et al. 1996, Karatzaferi et al. 1999). Phosphate stores are as well resynthesised quickly, as indicated by a half time of 30-40 seconds for CP stores (Tesch et al. 1986) and completely during next 2-5 minutes (Häkkinen 1990). For example, in the study of Häkkinen (1993) a three-minute resting interval between sets was enough to keep ATP and CP stores as the primary energy store in each set during very strenuous high resistance loading (20*1*100% of 1RM).

The recovering of glycogen stores begins after the exercise loading and continues the following 48 hours. Immediately after carbohydrate depleting exercise (i.e. prolonged

running), there is a short window of opportunity (perhaps up to 2 h) when muscle glycogen storage capacity has been enhanced substantially (Ivy et al. 1988). Therefore, recovery rate of glycogen stores depends on received food intake during recovery and the magnitude and duration of completed exercise (Häkkinen 1990). Figure 15 represents enhanced muscle glycogen resynthesis following by immediaty consume of carbohydrate after 70 min of cycling (Ivy et al. 1988).

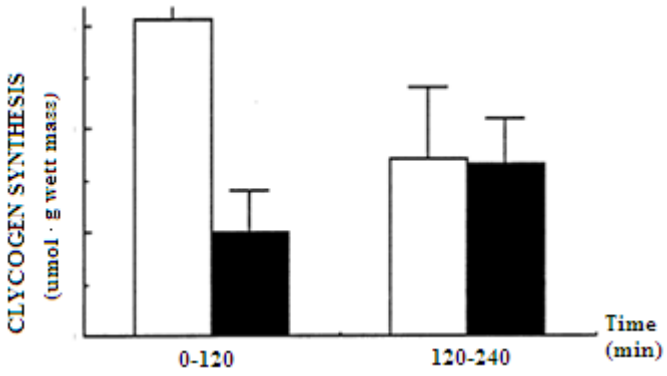

FIGURE 15. Muscle glycogen resynthesis during the first few hours when CHO ingested immediately (white) or 2 hours after (black) glycogen-depleting exercise (modified from Ivy et al. 1988).

In addition, many endurance studies have focused on the post-exercise decrease in blood lactate concentration as a measure of recovery (Cairns 2006, Toubekis et al. 2008). In the study of Skof & Strojnik (2005), intensive aerobic running increased blood lactate level (5.9 mmol/l) returned to its pre-exercise value within 60 minutes. That is in line with the finding that halflife of lactate lasts approximately 15-25 minutes after exercise, independent of total accumulation. Because lactate accumulation is dependent in turn on the type and amount of loading and duration of the work and recovery interval (Häkkinen 1993, Price & Moss 2007), it can be increased to a very high level after high intensity strength exercises, but usually returns to the pre-exercise value during 60-90 minutes of recovery (Ahtiainen et al. 2003, Senna et al. 2008). In the study of Tesch et al. (1986) the blood lactate increased up to 13.3 ± 2.7 mmol/l and it was still elevated after 40 minutes of recovery (figure 16).

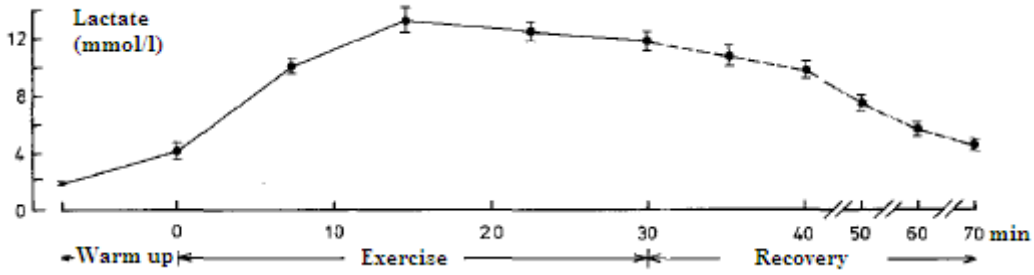

FIGURE 16. Blood lactate concentrations before, during and after heavy resistance exercise (modified from Tesch et al. 1986).

However, although blood lactate usually returns to resting levels approximately 1 hour after cessation of exercise, the strength level has been shown to be reduced in a much longer period of time in many studies regarding combined exercise (Sporer & Wenger 2003, Leveritt & Abernethy 1999). Therefore, Leveritt et al. (1999) suggested that it is unlikely that increased lactate production would be the main fatigue mechanism involved in combined exercise induced acute fatigue.

4.3 Recovery of serum hormone concentration

Previous studies have shown that heavy resistance, hypertrophic and long-term running exercises elicited acute hormonal responses recover differentially. In the study of Kraemer & Ratamess (2005), anabolic hormones such as testosterone and growth hormones were elevated during 15-30 minutes of post-resistance exercise. Whereas, two high intensity training sessions (performed the same day) increased serum total and free testosterone and cortisol levels and they recovered during the following hour (Häkkinen et al. 1988b). Nevertheless, Ahtiainen et al. (2003) suggest that if the loading is high enough the concentration of anabolic hormones can be increased even in the second morning after heavy resistance loading, which may be a compensation mechanism of the hormonal system against the exercise-induced stress.

In Schumann's (2011) study (figure 17), combined exercise did not result significant changes in serum total testosterone immediately after exercise but the total and free testosterone were significantly increased 24h and 48h after combined exercise when endurance was performed before strength exercise. Inversely, testosterone levels were slightly decreased 24h and 48h after combined exercise when strength exercise was performed first and endurance exercise after (Schumann 2011, 53).

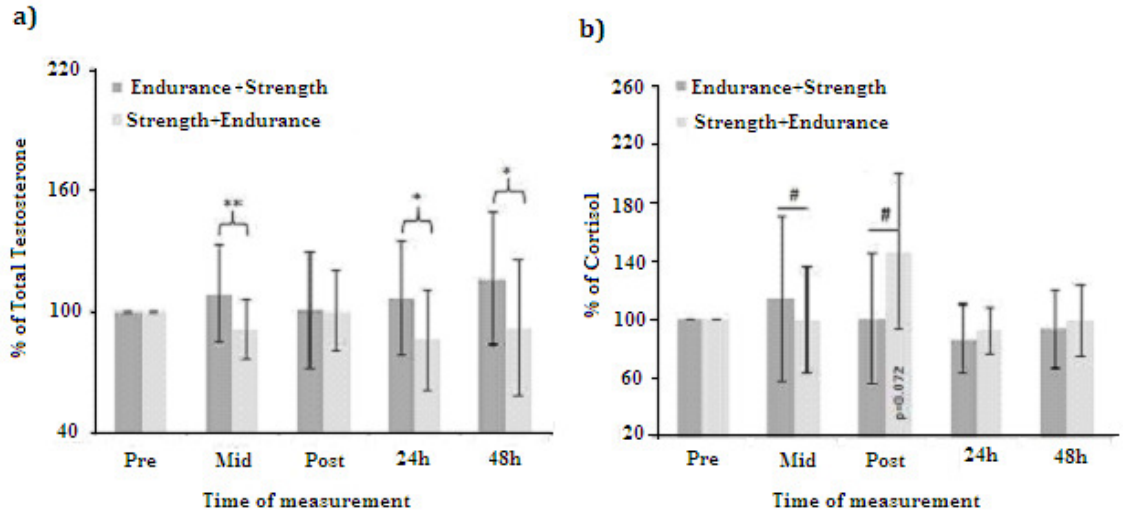

FIGURE 16. Combined exercise induced a) testosterone and b) cortisol responses of endurance athletes and comparison of endurance + strength and strength + endurance protocols possible order effect (modified from Schuman 2011, 53, 56).

Oppositely, the cortisol levels increased in both protocols after endurance running part of combined exercise (Schumann 2011, 50). The finding about increase of catabolic condition is supported by several studies regarding endurance exercise induced endocrinal responses. In the study of Kuoppasalmi et al. (1980), the intense long-term run significantly decreased mean plasma testosterone, which remained depressed up to 3 h after the end of the exercise. Similarly in the study of Trembley et al. (2005), testosterone showed a trend for a steady decline for the next 3 h of recovery after the long duration running. According to Ahtiainen et al. (2003), cortisol contributes to maintain sufficient rates of glycogen synthesis, protein turnover and supply of protein synthesis by amino acids in the post-exercise recovery period.

5 COMPARING ACTIVE AND PASSIVE RECOVERY

Several methods have been developed to enhance recovery from previous strength and endurance exercise bouts including active and passive recovery modes (Barnett 2006, Toubekis et al. 2008). Passive recovery is typically performed in order to reduce resting metabolic needs (Harper et al. 2008) whereas active recovery techniques enhances the circulation and metabolic waste removal from muscle and thereby facilitates recovery (Bogdanis et al. 1996, Kawabata et al. 2000, Siegler et al. 2006, Castagna et al. 2008, Thevenet et al. 2008). Several studies comparing active and passive recovery have reported that total blood lactate and muscle lactate concentration following by active recovery has been lower (Coffey et al. 2004, Greenwood et al. 2008, Bangsbo et al 1994), whereas, cardiovascular response (heart rate) have been higher in active recovery compared to passive recovery due to greater muscular engagement (Chrisafulli et al. 2003).

For example, Corder et al. (2000) investigated the effects of passive and active recovery during a resistance training workout and results showed active recovery (pedalling at 25% of lactate threshold) to be the most effective for reducing lactate and improving following squat performance. In addition, the results of Bogdanis et al. (1996) showed that active recovery enhanced not only lactate disappearance but also power output in following performance of repeated sprints. The explaining mechanism might be Mainwood & Renaud's (1985) finding of pH level of extracellular fluid affecting the recovery rate of force production (figure 17). The intensity associated with the lactate threshold is thought to be the optimal recovery intensity, since it is associated with a speed that should promote maximal lactate disappearance without additional lactate accumulation (Greenwood et al. 2008). If intensity of active recovery is close to the lactate threshold, it results in significant increases in lactate concentration and reduce the time to exhaustion (Thevenet et al. 2008).

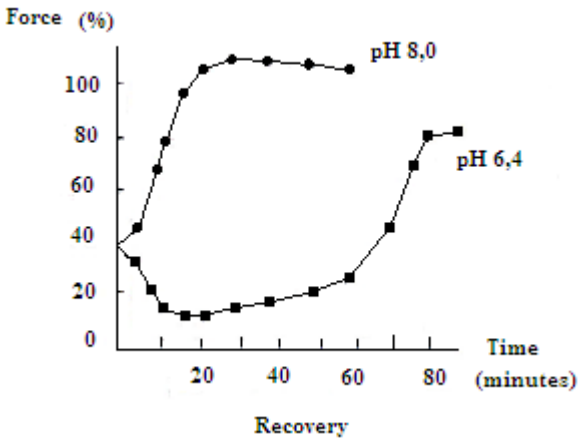

FIGURE 17. Recovery of force production after pH decreament (modified from Mainwood & Renaud 1985).

However, an almost equal number of studies have found little or no improvement in performance by implementing active recovery (Zainuddin et al. 2005, Cairns 2006, Thevenet et al. 2008, Toubekis et al. 2008). For example, Siegler et al. (2006) found that although blood acid-base recovery was affected by recovery mode (passive versus active), subsequent performance times (cycling to the exhaustion) remained similar. It is also hypothesized that increased metabolic rate during active recovery, despite the increase in oxygen uptake and aerobic energy system activation may counteract phosphocreatine and glycogen resynthesis (Barnett 2006). Therefore, employing active recovery may not be beneficial in all cases for performance improvements, and a more specific examination to identify any limitations is required (Toubekis et al. 2008).

If an individual is suffering from delayed-onset-muscle-soreness (DOMS), complete rest might be the optimal recovery mode being a common prescription for most musculoskeletal injuries, especially in the early stages of recovery (Zainuddin et al. 2005). Passive recovery can be performed by sleeping, resting or seating (Thevenet et al. 2008) and, therefore, minimizing the use of the injured tissue is believed to prevent further damage and promote the processes of repair and regeneration from eccentric-exercise-induced muscle damage (Zainuddin et al. 2005).

6 PURPOSE OF THE STUDY

The main purpose of the study was to determine the acute combined strength and endurance exercise induced fatigue responses on maximum isometric force production, muscle activity and work economy. In addition, the changes of blood lactate and heart rate were detected. The secondary focus was to compare active and passive recovery models to the neuromuscular and physiological recovery rate in men and to investigate how the individual fitness level of the subject affected the rate of fatigue and recovery.

6.1 Research objectives

1. Neuromuscular performance. To examine combined strength and endurance exercise induced acute neuromuscular responses on force production capacity, muscle activation and work economy.

2. Active and passive recovery. To compare the effects of active and passive recovery on force production capacity, blood lactate removal and heart rate dynamics after combined strength and endurance exercise.

3. Fitness level of the subject. To examine the effects of subjects' individual fitness levels on fatigue and recovery rate of neuromuscular performance after the combined strength and endurance exercise.

6.2 Hypotheses

Hypothesis 1. Combined strength and endurance exercise loading will provoke neuromuscular fatigue response characterized by decreases in maximal isometric strength (MVC), fast force production capacity (force-time curve) and muscle activation (EMG). In addition, the subject's work economy will decrease. (Tesch & Wright 1983, Häkkinen et al. 1988a, Häkkinen 1993, Häkkinen1994, Linnamo et al. 2000.)

Hypothesis 2. Active recovery facilitates recovery by particularly increased blood flow and thereby enhances the metabolic waste removal and recovery rate of maximal isometric force (MVC) compared to passive recovery but interferes therefore the recovery rate of heart rate (HR). (Bogdanis et al. 1996, Corder et al. 2000, Barnett 2006.)

Hypothesis 3. The better the cardiovascular (VO_2max) and neuromuscular performance (leg press 1-RM) of the subject in the pre-measurements, the lesser the decrements of force (MVC) and rise of heart rate (HR) and the faster the recovery of strength (MVC) and heart rate (HR) after the combined strength and endurance loading will be. (Tesch & Wright 1983, Häkkinen 1990, Viitasalo & Lahtinen 1998.)

7 METHODS

7.1 Subjects

The subjects included 16 randomly selected Finnish male reservists in good health, aged between 23 and 37 years (mean 27 ± 4). The experimental group (n=16) was divided into the active recovery group (AR, n=8) and the passive recovery group (PR, n=8). The variables of the preprotocol measurements were normally distributed (except for the age of the subjects) and there were no significant differences (except for in body mass) between the groups in subjects' characteristics (Table 1). All subjects were informed of the risks and benefits of the study prior to completing a written informed consent (appendix 1). They were also asked to avoid vigorous exercise, consumption of alcohol and caffeine and the use of tobacco 4 h prior to each measurement day. In addition the subjects came in a fasted (12h) state to the measurements of each testing day. They performed pre-protocol control measurements, pre-measurements, combined strength and endurance loading, active or passive recovery and follow-up measurements.

TABLE 1. Subjects characteristics measured in pre-protocol control day (Mean ± SD) in the active (AR) and passive (PR) recovery groups.

Group	**AR (n=8)**	**PR (n=8)**
Age (yr)	27 ± 4	27 ± 4
Height (cm)	182 ± 5	176 ± 7
Body mass (kg)	84 ± 9	71 ± 8
BMI ($kg \cdot m^{-2}$)	25 ± 3	23 ± 1
Body fat (%)	17 ± 6	14 ± 4
VO_{2max} (ml/kg/min)	57 ± 9	59 ± 6
1-RM (kg)	181 ± 20	159 ± 29
HRrest (bpm)	63 ± 12	59 ± 7
HRmax (bpm)	196 ± 6	196 ± 9

7.2 Study design

The overall study design is shown in figure 18 and the detailed measurement timetable is displayed in appendix 2. The study was conducted according to the Declaration of Helsinki and was approved by the Ethical Committees at the University of Jyväskylä and the Central Finland Health Care District, as well as the Surgeon General of the Finnish Defence Forces. All measurements were carried out in the laboratories of the Department of Biology of Physical Activity at the University of Jyväskylä.

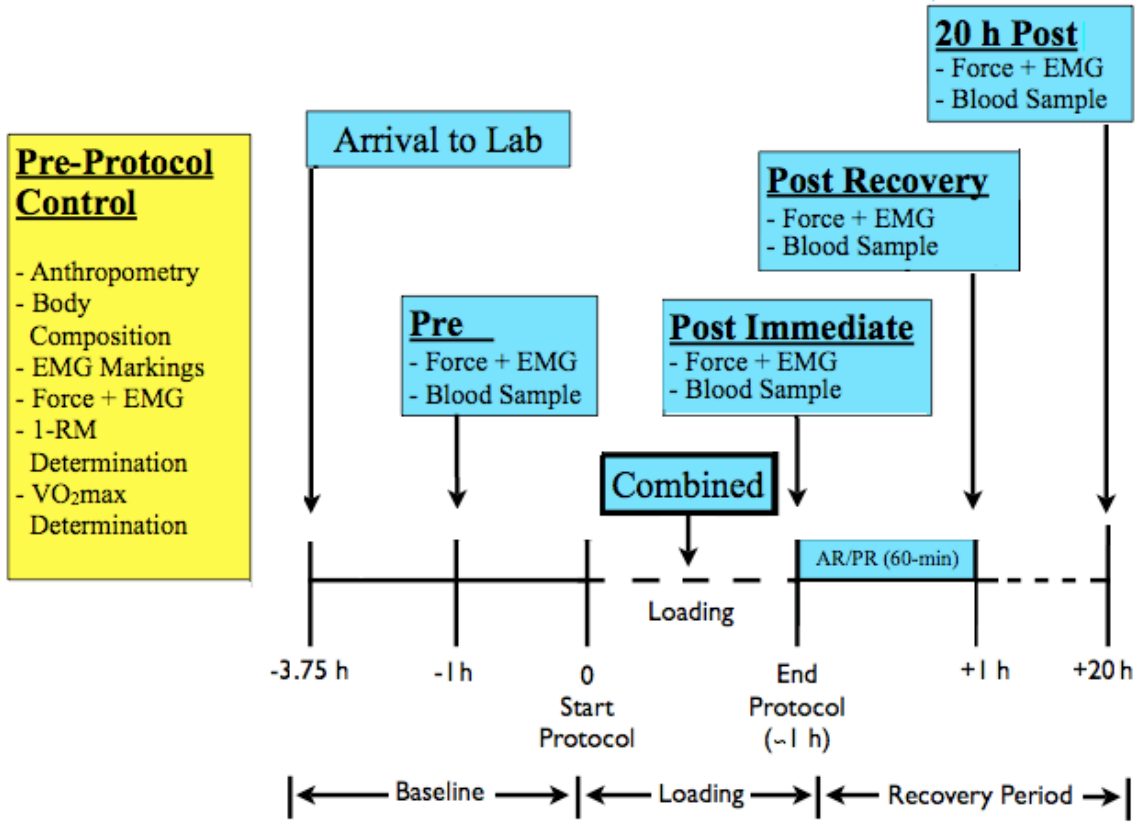

FIGURE 18. A simplified study design.

Pre-protocol control measurements were completed 14 ± 10 days prior to the loading day. Measurements consisted of anthropometry (height, weight, the body composition) measurements, markings of electrode positions, familiarization force measurements and the determinations of endurance (VO_{2max}) and strength (1-RM) capacities (figure 18). Also the maximum and the rest heart rate (HR) of subjects' were detected.

Loading day control measurements. The subjects were acting as their own controllers and, therefore, the control measurements were done before the onset of loading. The loading day control force measurements were taken one hour after consuming an energy bar with water, approximately one hour before the loading. The loading day control measurement procedure details are explained in appendix 2.

The combined strength and endurance loading was performed 3-36 days (average 14 ± 10 days) following the control measurements. The loading protocol can be seen in appendix 3. The loading started at approximately the same time of the day (11.00) for all subjects (which was about two hours after consuming an energy bar (700 kJ/170 kcal, proteins 7 g, carbohydrates 21g, fats 2.5g)) and lasted for 55 minutes. During the loading, the subjects were wearing special military equipment; with a total extra load of 16 kg (aproximately 20 % of subjects' body mass). Therefore, subjects were weighed prior to the loading by the digital scale (accuracy 0.1 kg) without and with the whole experimental gear in order to assess the extra load. The military gear included a weighted vest, a helmet, a rifle, and the army boots (figure 19).

FIGURE 19. The military simulating combined strength and endurance loading contained treadmill run/walk with the military gear.

The loading consisted of 30-minutes treadmill run/walk with the military gear and 5 sets of 10 repetitions dynamic bilateral leg press exercise using 70% of their pre-determined 1-RM with 2 minutes rest between sets. The order of exercises was 5-min at 4,5 km/h, 5-min at 7,0 km/h, 2 sets of 10 repetitions at 70% 1-RM, 10-min at 4,5 km/h, 1 set of 10 repetitions at 70% 1-RM, 5-min at 7,0 km/h, 5-min at 4,5 km/h and 2 sets of 10 repetitions at 70% 1-RM. Breath-by-breath oxygen consumption, HR and RPE were measured throughout the marching on a motor-driven treadmill. The subjects were allowed to take of the rifle, vest and the helmet and to change the military boots for their own sneakers while performed leg press exercise. The load of dynamic leg press was assessed so that the subject could barely lift all 10 reps by himself. The assistance was given by the assistants during the concentric phase of the leg press, when necessary. (Figure 20.)

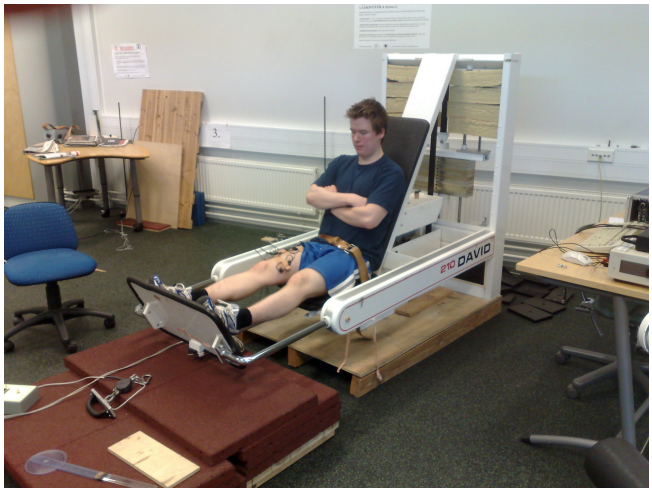

FIGURE 20. Leg press exercise consisted of 5*10*70% of subjects' pre-determined 1-RM with 2 minutes rest between sets.

Recovery protocols. After the loading, subjects completed either active (AR n=8) or passive (PR n=8) recovery for 60-minutes. Active recovery started 11 ± 3 minutes after the end of the loading and followed an identical recovery procedure which consisted of 5 sets of 10 repetitions at 30% of subject 1-RM, including 5 minutes of rest between sets and 30 minutes of treadmill walking at walking speed of 3.0 km·h-1 for all subjects using 5-minutes intervals (5-min on: 5-min rest) (appendix 4). The passive recovery

started also 11 ± 3 minutes after the end of the loading and was done by resting in a chair for 60 minutes. The HR recovery was measured throughout the recovery period (average of every 5-min) in both (AR and PR) groups.

Follow-up measurements (MVC and blood lactate) were completed immediately (2±1 minutes) after the loading for analyse of subjects' fatigue responses. About an hour later, immediately after the end of the recovery protocol, the measurements were repeated in order to measure the recovery. The 20-hour post measurements were performed in the next morning followed by the same procedures as on the testing day.

7.3 Measurements

Anthropometrics. The subject's anthropometrical data are shown in table 1. Standing height was measured by a wallmounted inelastic plastic tape (accuracy 0.1 cm), subjects standing barefoot. Bioelectrical impedance analysis (BIA; InBody 720, Seoul, South Korea) was used for the assessment of body mass. Subjects wore only light sports clothes and no shoes. Body mass index (BMI) was calculated by dividing body mass in kilograms by the square of height in meters (kg/m^2). The percentage of body fat was estimated according to the method of Jackson & Pollock (2004). Skin fold thickness measurements were taken twice at seven sites (chest, *axilla*, *triceps*, *subscapular*, *suprailiac, abdomen*, and thigh) on the right side of the body using a skin fold caliper (John Bull Skin Fold Caliper, British Indicators, LTD, UK). The third skin fold measurement was taken only if the first two scores differed more than 0.2 mm. The closest two measurements were averaged by ± 0.4 mm.

Blood lactate. Capillary blood samples were taken from the fingertip by a qualified lab technician and used for determination of blood lactate. Analyses were performed using Biosen lactate analyzer (S_line Lab+, EKF Diagnostic, Magdeburg, Germany).

Heart rate. The resting heart rate was measured during the 20-minutes laying in the pre-protocol control measurements, however, the first and the last 5 min were excluded and the lowest minute from the rest of the 10-min data was used for analysis. The maximum HR (HR_{max}) was the highest HR value reached during the graded treadmill test in the pre-protocol control measurements. Heart rate (HR) was measured during the loading and recovery by Suunto t6 heart rate monitor (Suunto Inc., Vantaa, Finland) and chest strap, which was moisted so that the contact and reading would be reliable. The HR was also measured in particular timepoints throughout the day (average of every 5-min in HR_{pre}, HR_{post1h} and $HR_{post20h}$ and average of 1 minute in $HR_{postload}$). All the heart rate data was analyzed by KubiosHRV 2.0 software (Kuopio, Finland).

Maximum aerobic capacity. Maximum aerobic capacity was determined with a graded running test on a motor-driven treadmill (OJK-1, Telineyhtymä, Kotka, Finland). The running speed was set between 9.5-12 $km \cdot h^{-1}$. The graded oxygen uptake (VO_{2max}) test was preceded by 5-min warm-up, using 5.0 $km \cdot h^{-1}$ walking speed for the first two minutes and 8.0 $km \cdot h^{-1}$ for the last three minutes of the warm-up on level grade. After the warm-up, the individual constant speed was set, chosen according to the endurance training history. After the first 0% grade stage of two minutes, the grade of the treadmill was increased by two degrees after every two minute stage. All subjects were verbally encouraged to continue until maximal exhaustion. O_{2max} was defined as the highest 30-s O_2 during the test. O_{2max} was reached when 3 of 4 criteria were met: (1) the 60-s O_2 value reached plateau or started to decrease, (2) respiratory exchange ratio (RER) was over 1.13, (3) the HR was 10 beats from the predicted HRmax (predicted HRmax = 220 - age) or failed to increase with added workload, and (4) RPE value was greater than 17 (ACSM 2006). The rate of perceived exertion (RPE) in every 2 minutes was assessed by the Borg's scale (6-20), established on Borg's psychological and physiological studies (Borg 1982).

During a 5-min cool-down period, the walking speed was individually chosen between 3.0-4.0 $km \cdot h^{-1}$. The breath-by-breath oxygen consumption (O_2) was measured during the whole test using a portable gas analyzer (Oxycon Pro Jaeger, VIASYS Healthcare GmbH, 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 CO_2 4.11 ± 0.08% and O_2 15.6 ± 0.3%. All subjects wore flow-by face masks with dead space of 40 ml (Hans Rudolph, Kansas City, MO, USA). The mask covered the nose and the mouth, and it was carefully fitted in order to prevent air leakage.

One-repetition-maximum dynamic leg extension. Dynamic maximal strength of leg extensors (conducted in order to determine the load for the loading-day) was measured in a seated position using the bilateral horizontal leg press on the David 210 dynamometer (David Sports Ltd., Helsinki, Finland). Prior to attempting 1RM, subjects completed a warm-up consisting of 3-6 x 70% RM and 4-5 x 80 – 85% 1 RM with two minutes of rest between the sets. Following this warm up, no more than 5 attempts to reach 1 RM were made. Leg extension action started from a knee angle of approximately 60 degrees. Subjects were instructed to keep their hands crossed on their chest 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 1 RM. (Häkkinen et al. 2003.)

Work economy. Work economy during the loading was assessed as the oxygen consumption during the two identical 5-min stages; the first and last one. The O_2 values are expressed in relation to the real body mass during the loading thus extra weight included ($ml \cdot min^{-1} \cdot kg^{-1}$) and the relative intensity of the loading at a certain stage was calculated against the adjusted O_{2max} value, since the maximal O_2 uptake was measured without the extra load. The breath-by-breath data was used to evaluate the walking economy.

Maximal isometric leg extension force. Bilateral isometric leg extension force (MVC) assessments were performed using a horizontal electromechanical dynamometer (Department of Biology of Physical Activity, University of Jyväskylä, Jyväskylä, Finland). The subjects were in a seated position with a knee angle of 107º which was

individually set to each subject for every measurement point. The arms were kept crossed on the chest. (Figure 21.) The 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 and to keep constant contact with the seat and backrest throughout each measurement trial. They were also verbally encouraged to give their real maximum.

All subjects had three trials with one minute rest between, and the best performance was used for further analysis. Maximal force was accepted when the difference between two trials did not exceed 5%. Before the morning measurements five additional sub-maximal warm-up trials (3 x ~50% and 2 x ~85%) were performed by the subjects before the maximal contraction recordings. The force signal was low pass filtered (20 Hz) and analyzed (Signal software Version 4.04, Cambridge Electronic Design Ltd, Cambridge, UK). All of the maximum isometric leg extension force (MVC) data was analyzed by customized script (Signal 2.16, CED, UK) and all values are given in Newtons (N). (Häkkinen1993.)

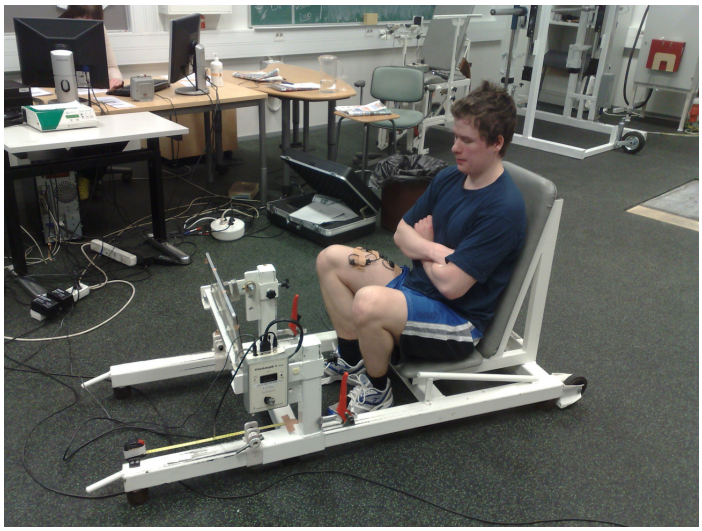

FIGURE 21. Before the measurement and during the one minute rest periods between the sets subjects could keep their knees flexed whereas during the actual leg extension action the knee angle was set to 107º.

Electromyography. Electromyographic activity (EMG) was recorded from the agonist muscles vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) of the right leg during the maximal isometric action. Bipolar surface electrodes (Beckman miniature-sized skin electrodes 650437, Schiller Park, Illinois) with 20-mm interelectrode distance were employed. The electrodes were placed longitudinally over the muscle belly. The positions of the electrodes were marked on the skin by small ink dots to ensure the same electrode positioning in each test during the experimental period. EMG signals were recorded by Signal 2.15 (Cambridge Electronic Design Ltd. 1997-2004 and Noraxon, Telemyo 2400R, USA, Inc.), full-wave rectified, integrated (iEMG), and time normalized. The activity (AEMG, iEMG) of the VL, VM and RF muscles was averaged and analysed in the maximal force phase (100ms and 1500-2500ms) of the isometric muscle actions. (Häkkinen et al. 2003.)

7.4 Statistical analyses

Microsoft Office Excel 2007 conventional statistical methods were used for the calculations of means, standard deviations (SD) (and standard errors SE). The results are presented as group mean ± standard deviation (SD) and/or standard error (SE). Shapiro Wilks test and histograms were utilized to examine the distribution of the variables. Between the groups, differences (active and passive recovery groups) were analysed using independent-samples T-test. Within each group, differences were analysed using repeated measures ANOVA with 4 levels (Pre, Postload, Post1h, Post20h). The relationships between different variables were examined with the help of the regression analysis and Pearson's correlation coefficient.The significance was set at *$p<0.05$, ** $p< 0.01$ and ***$p<0.001$. Statistical analysis was completed with PASW 19.0 (SPSS Inc., Chicago, IL, USA).

8 RESULTS

8.1 Acute neuromuscular responses

8.1.1 Maximal isometric force production

Maximal isometric force of legs (MVC) decreased significantly ($p < 0.001$) during combined strength and endurance exercise loading. The mean (n=16) decrease of force was 416 ± 313 N (14.2 ± 9.5 %) from the pre-exercise value (2937 N). (Table 2.) There were no significant differences between the active and passive recovery groups in the magnitude of strength loss.

TABLE 2. Maximal isometric force production (N) of active recovery (AR), passive recovery (PR) and whole experimental group (n=16) measured before and after the combined loading.

Measurement	**Pre**			**Postload**			**Post1h**			**Post20h**		
Group	AR	PR	n=16	AR	PR	n=16	AR	PR	n=16	AR	PR	n=16
Mean	3009	2864	2937	2595	2447	2521	2782	2580	2681	3045	2846	2946
SD	602	1013	808	512	815	662	713	841	760	805	855	808

The decreased force level (MVC) recovered 160 ± 294 N (6.3 ± 8.5 %) (n=16) during the recovery period from immediately after the loading measured value (2521 N). MVC increased 187 ± 262 N (5.3 ± 8.4 %) in the AR group (n=8) and 113 ± 339 N (4.5 ± 13.5 %) in the PR group (n=8) and there was no significant difference between the active and passive recovery groups (figure 22). On the contrary, recovery of force showed a positive (r=0.814, p=0.01) correlation with recovery of subjects' blood lactate level. Next morning, 20 hours after the combined loading, MVC was significantly increased from the post-load ($p < 0.001$) and post1h ($p < 0.05$) values and even slightly (9 ± 277 N, 0 ± 10 %, n=16) above the pre-exercise value. There was no significant difference of force recovery rate identified between the active and passive recovery groups. (Figure 23).

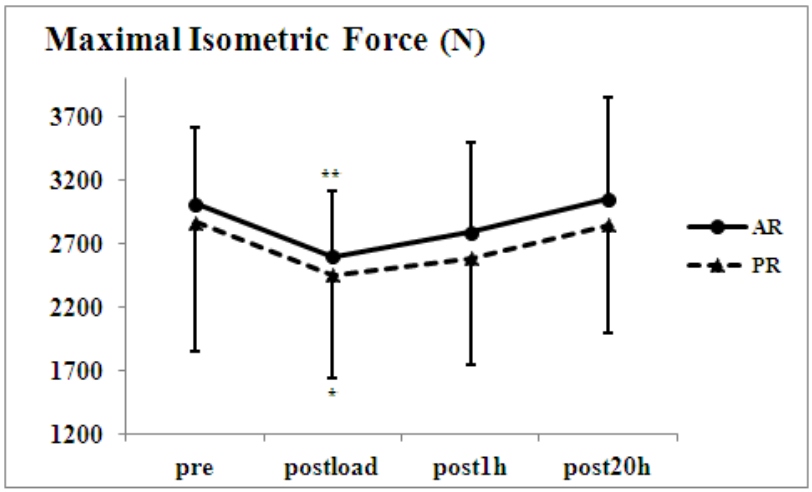

FIGURE 22. Mean (± SD) maximal isometric force (MVC) before (pre), immediately after the loading (postload), immediately after the recovery (post1h) and next morning (post20h) in the active (AR, n=8) and passive (PR, n=8) recovery groups. Significant difference compared to the pre-exercise value (**= $p < 0.01$, * = $p < 0.05$).

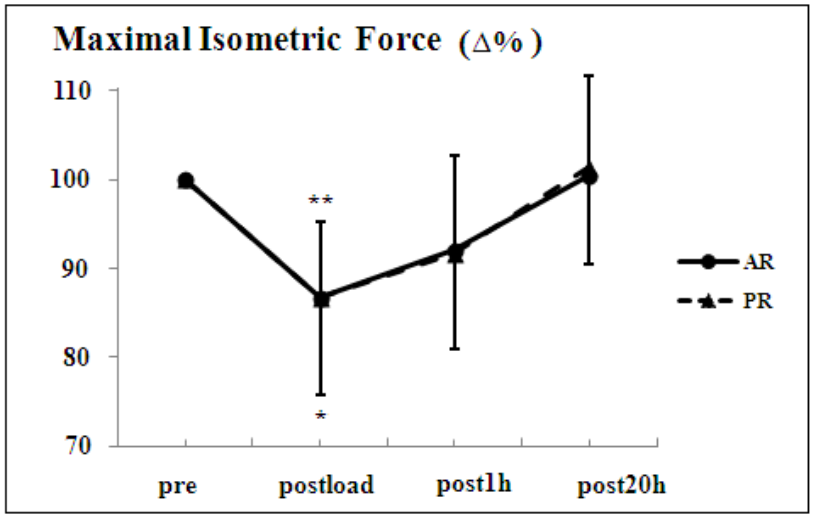

FIGURE 23. Relative (± SD) change of maximal isometric force (MVC) before (pre), immediately after the loading (postload), immediately after the recovery (post1h) and next morning (post20h) in the active (AR, n=8) and passive (PR, n=8) recovery groups. Significant difference compared to pre-exercise value (**= $p < 0.01$, * = $p < 0.05$).

8.1.2 EMG-activity

Significant decreases ($p < 0.05$ - $p < 0.01$) occured in the averaged EMG of the (1500-2500 ms) isometric action due to the combined loading protocol in both groups. (Table 4.) The mean (n=15) AEMG of the vastus lateralis (VL) decreased ($p<0.01$) 36 ±44 % from its pre-exercise value (in AR, n=7, $p < 0.05$, 57 ± 28% and in PR, n=8, $p<0.01$, 30 ± 56%). The mean (n=14) activation of vastus medialis (VM) decreased 22 ± 43% (in AR, n=6, 18 ± 37 % and in PR, n=8, 24 ± 50%). The mean (n=14) AEMG of rectus femoris (RF) decreased ($p<0.05$) 21±59 % (26 ± 24 % in AR, n=8 and 14 ± 90 % in PR, n=6) during the loading. There were no significant differences between the active and passive recovery groups.

TABLE 4. Averaged EMG (mean±SD mV) before (pre), immediately after the loading (postload), immediately after the recovery (post1h) and next morning (post20h).

Measurement	**Pre**			**Postload**			**Post1h**			**Post20h**		
Muscle	VL	VM	RF	VL	VM	RF	VL	VM	RF	VL	VM	RF
Mean	0,20	0,34	0,14	0,12	0,24	0,08	0,17	0,27	0,11	0,12	0,27	0,18
SD	0,12	0,26	0,16	0,10	0,24	0,12	0,16	0,32	0,15	0,11	0,31	0,23
N	15	14	14	16	16	16	16	14	16	15	13	15

Statistically significant changes did not occur in AEMG activity during the recovery ($post_{1h,}$ $post_{20h}$) (figures 24 and 25) period between the measurements, whereas, between the recovery groups the significant ($p<0.05$) differences was found in vastus lateralis ($post_{1h,}$ $post_{20h}$). The muscle activity of VL (n=15) increased 4 ± 54 % (in AR, n=7, 11 ± 27% decrease and in PR, n=8, 15 ± 76% increase) during the 60-minutes recovery but decreased thereafter 25 ± 94% (in AR, n=7, 1 ± 23 % and in PR, n=8, 48 ± 126 %). Whereas the activity of VM (n=12) decreased 2 ± 54 % (in AR, n=6, 0 ± 61 % and in PR, n=6, 5 ± 52 %) but increased thereafter 14 ± 60 % (n=11) (in AR, n=5, 3 ± 20 % decrease and in PR, n=7, 27 ± 78 % increase). The muscle activity of RF (n=12) decreased 12 ± 42 % (in AR, n=6, 5 ± 48 % increase and in PR, n=6, 39 ± 19 % decrease) during the recovery and turned to 51 ± 85 % (n=12) increase thereafter (in AR, n=5, 52 ± 92 % and in PR, n=7, 52 ± 79 %).

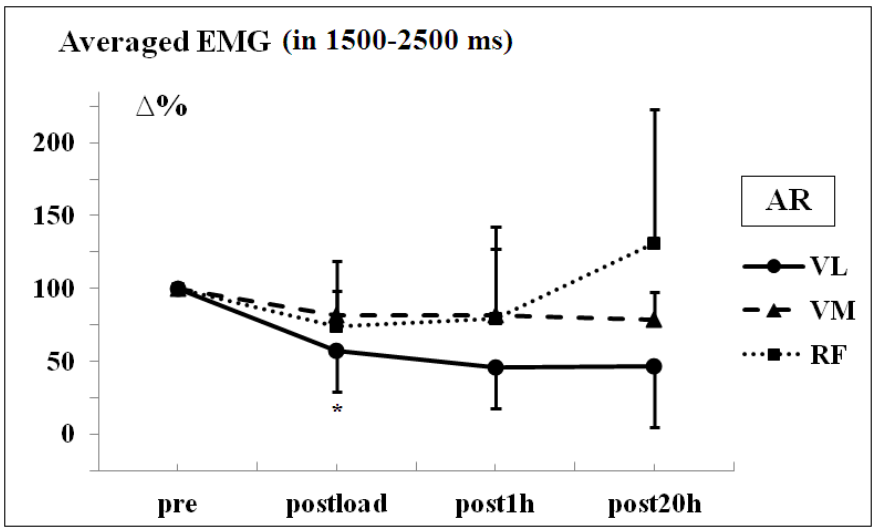

FIGURE 24. Averaged EMG in 1500-2500ms (mean±SD) of vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) in the active recovery (AR, n=8) group before (pre) and after (postload) the combined loading and recovery (post1h, post20h) in reservists men. Significantly different from the pre-exercise value (* = p<0.05).

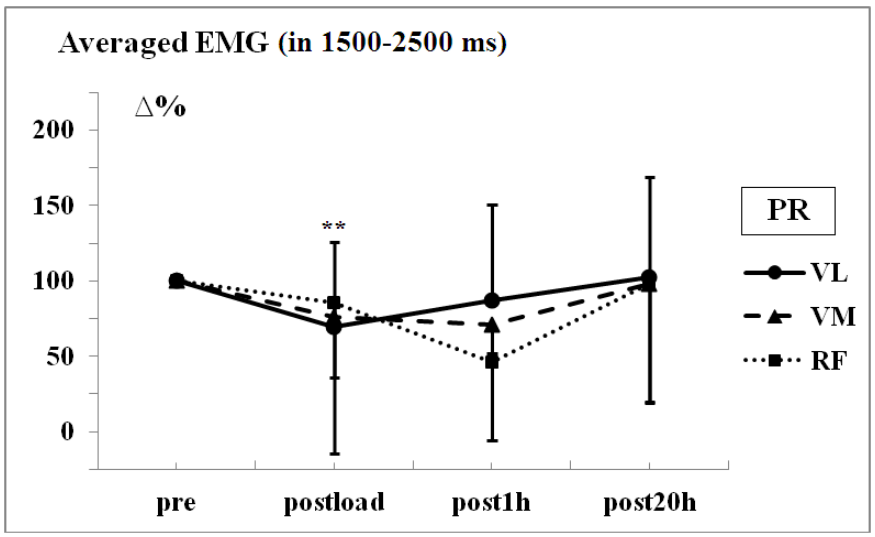

FIGURE 25. Averaged EMG in 1500-2500ms (mean±SD) of vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) in the active recovery (AR) group before (pre) and after (postload) the combined loading and recovery (post1h, post20h) in reservists men (n=8). Significantly different from the pre-exercise value (** = p<0.01).

There were also significant (p<0.05) changes in integrated EMG (in 100ms) due to combined strength and endurance exercise loading. (Figures 26-27.)

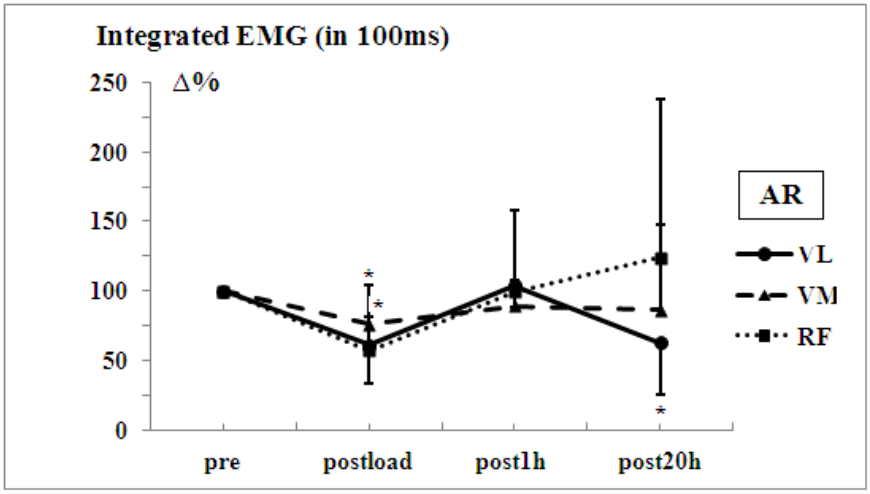

FIGURE 26. Integrated EMG in 100ms (mean±SD) of vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) in the active recovery (AR) group before (pre), immediately after the loading (postload), immediately after the recovery (post1h) and next morning (post20h) in reservists men. Significantly different from the pre-exercise value (* = p<0.05).

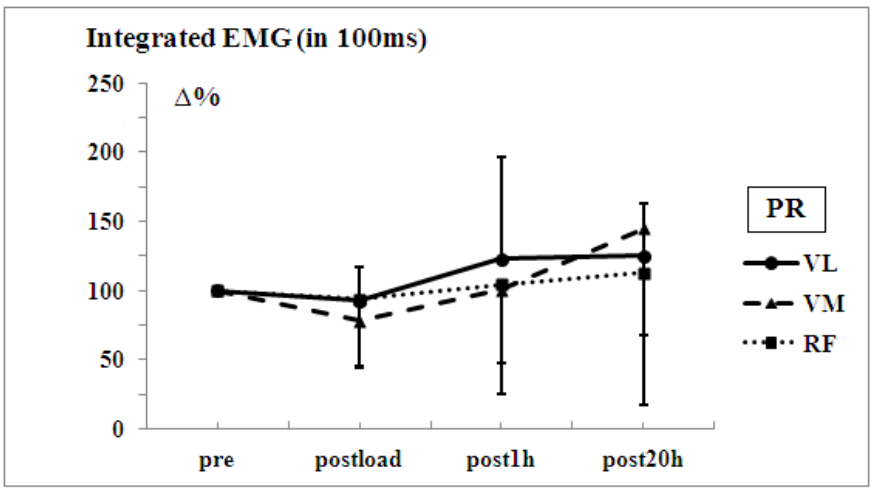

FIGURE 27. Integrated EMG in 100ms (mean±SD) of vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) in the passive recovery (PR) group before (pre), immediately after the loading (postload), immediately after the recovery (post1h) and next morning (post20h) in reservists men.

The mean (n=16) decrease of iEMG of vastus lateralis (VL) was 28 ± 35% (p<0.05) from its pre-exercise value (48 ± 34% in AR, n=8, and 8 ± 25% in PR, n=8). The mean (n=16) iEMG-decrease (in 100 ms) of vastus medialis (VM) was 23 ± 29% (24 ± 28 % in AR, n=8, and 22 ± 33% in PR, n=8) and in rectus femoris (RF, n=16) 25 ± 42 % from its pre-exercise value (43 ± 24 % in AR, n=8, and 6 ± 50% in PR, n=8).

The level of iEMG (in 100 ms) recovered to the pre-exercise level during the 60-minutes recovery period. The muscle activity of VL (n=14) increased (p<0.05) 34 ± 60 % (in AR, n=7, (p<0.05) 37 ± 42 % and in PR, n=7, 31 ± 74 %) during the 60-minutes recovery but decreased thereafter 21 ± 57% (in AR, n=7, 44 ± 43 % decrease and in PR, n=7, 2 ± 38 % of increase). The activity of VM (n=15) increased continuously 39 ± 78 % (in AR, n=7, 10 ± 64 % decrease and in PR, n=8 (p<0.05) 44 ± 118 % increase) being above its pre-exercise value next morning. Also the mean iEMG (in 100 ms) of RF (n=16) increased significantly (p<0.05) from post-load values for next morning. The increase was 25 ± 57 % (in AR, n=8, 42 ± 59 % and in PR, n=8, 5 ± 58 %) during the recovery and continued 20 ± 80 % thereafter (in AR 25 ± 113 % and in PR 15 ± 43 %). There were no significant differences between the active and passive recovery groups.

8.1.3 Force-time curve

The force-time curve of the leg extension shifted significantly (p<0.05-0.01) to the lower level due to combined loading in both groups. After the loading the force time-curve differed significantly in every timepoint from the beginning (0-100ms p<0.01) to the end (400-500 ms p<0.01) (n=16) comparing to values measured before the loading. (Figures 28-29.)

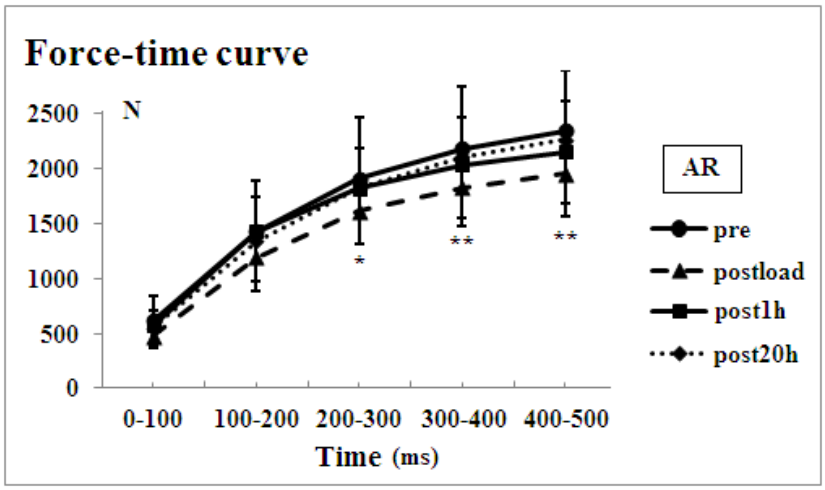

FIGURE 28. Force-time curves (mean ± SD) of the active (AR, n=8) recovery group's leg extension MVC before (pre), immediately after the loading (postload), immediately after the recovery (post1h) and next morning (post20h) in reservists men. Significant difference compared to pre-exercise value (**= $p < 0.01$, * = $p < 0.05$).

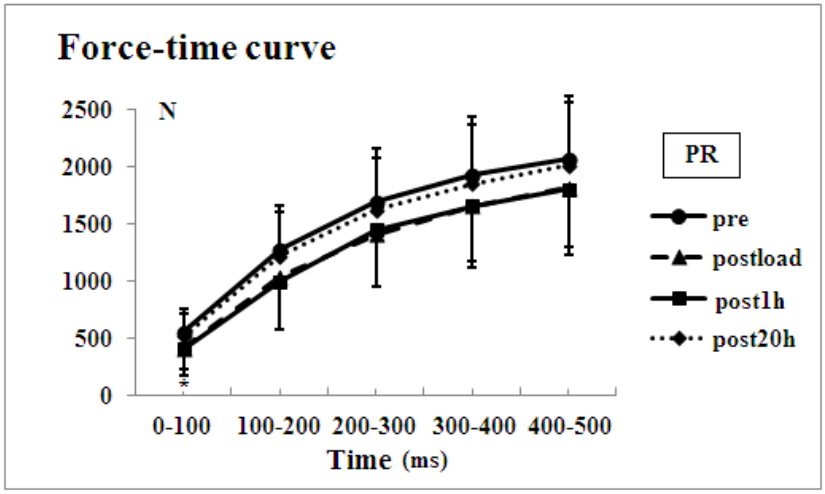

FIGURE 29. Force-time curves (mean ± SD) of the passive (PR, n=8) recovery group's leg extension MVC before (pre), immediately after the loading (postload), immediately after the recovery (post1h) and next morning (post20h) in reservists men. Significant difference compared to pre-exercise value (* = $p < 0.05$).

The mean (n=16) force decreased (p<0.01) 140 ± 157 N (141 ± 177 N in AR, n=8, and 139 ± 147 N, p<0.05 in PR, n=8) between 0-100 ms, (p<0.05) 235±363 N (232 ± 421 N in AR, n=8 and 237 ± 325 N in PR, n=8) between 100-200 ms, (p<0.01) 290 ± 331 N (298 ± 297 N, p<0.05 in AR, n=8 and 282 ± 383 N in PR, n=8) between 200-300 ms, (p<0.01) 312 ± 365 N (272 ± 430 N, p<0.01 in AR, n=8 and 352 ± 311 N in PR, n=8) between 300-400 ms and (p<0.01) 322 ± 369 N (392 ± 297 N, p<0.01 in AR, n=8 and 253 ± 439 N in PR, n=8) between 400-500 ms. There were no significant differences between the groups (AR vs PR) in any timepoint.

After the recovery period (n=16) force increased 72 ± 168 N between 0-100 ms (94 ± 213 N in AR, n=8 and 33 ± 112 N in PR, n=8), 142 ± 337 N between 100-200 ms (213 ± 411 N in AR, n=8 and 48 ± 233 N in PR, n=8) , 122 ± 404 N between 200-300 ms (207 ± 511 N in AR, n=8 and 36 ± 368 N in PR, n=8), 101 ± 491 N between 300-400 ms (219 ± 625 N in AR, n=8 and 0 ± 322 N in PR, n=8) and 90 ± 520 N (196 ± 657 N increase in AR, n=8 and 16 ± 351 N decrease in PR, n=8) between 400-500 ms.There were no statistically significant differences from the post-load values or between the groups (AR vs PR) in any timepoint.

Next morning ($post_{20h}$) (n=16) force still increased 23 ± 251 N between 0-100 ms (23 ± 335 N decrease in AR, n=8 and 72 ± 85 N increase in PR, n=8), 16 ± 590 N between 100-200 ms (90 ± 800 N decrease in AR, n=8 and 131 ± 139 N increase in PR, n=8), 80 ± 700 N (p<0.01) between 200-300 ms (2 ± 958 N decrease in AR, n=8 and 175 ± 200 N increase in PR, n=8), 127 ± 790 N (p<0.001) between 300-400 ms (77 ± 1101 N increase in AR, n=8 and 195 ± 198 N increase in PR, n=8). Between 400-500 ms (n=16) the mean force increased 271 ± 844 N (p<0.001) (114 ± 1186 N increase in AR, n=8 and 214 ± 187 N increase in PR, n=8).There were no significant differences between the groups (AR vs PR) in any timepoint.

8.2 Acute physiological responses

8.2.1 Heart rate

The absolute increases and reductions in HR values from the beginning to the end of the loading day were not significantly different between the groups. Before the loading the mean (n=16) resting heart rate (HR_{pre}) was 62 ± 8 bpm (in AR, n=8, 63 ± 8 bpm and in PR, n=8, 62 ± 7 bmp). During the loading HR increased significantly (p = 0.001) 49 ± 10 % to 120 ± 25 bpm (n=10) (114 ± 25 bpm in AR, n=4 and 126 ± 25 bpm in PR, n=6). Figure 30 presents how heart rate was significantly (p < 0.001) elevated during the whole 55 min period of loading in both active (AR) and passive (PR) recovery groups. The HR values differed significantly (p = 0.05) only at timepoint of (mean of) 5-10 min between AR and PR groups.

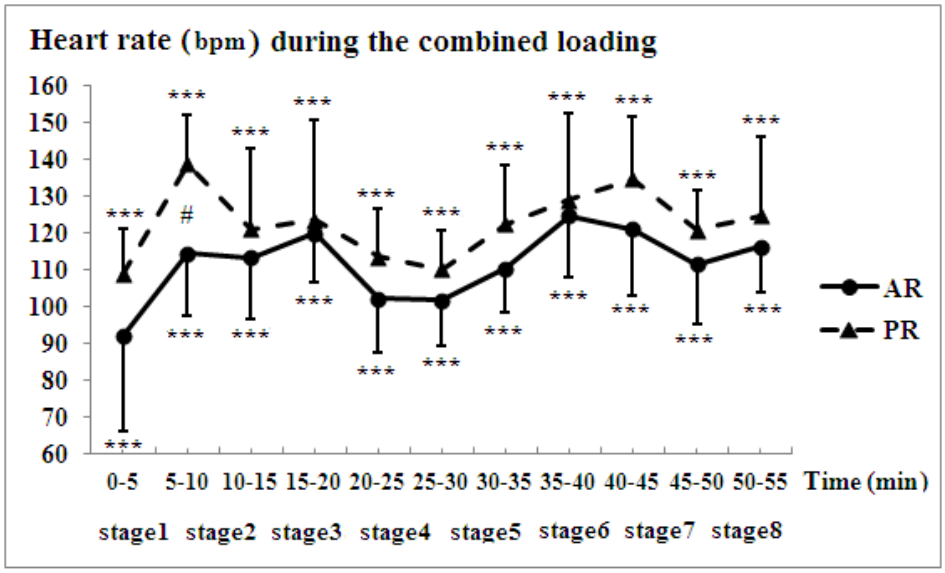

FIGURE 30. The mean (of 5min) heart rate (±SD) of the active (AR, n=4) and passive (PR, n=6) recovery groups during the combined loading. Significantly different (*** = p < 0.001) from the pre-exercise value. Significant difference (# = p < 0.05) between the groups.

During the following recovery period the mean (n=16) heart rate level decreased significantly (p=0.001) 59 ± 23 bpm (48.76 ± 13.79 %) (in AR, n=8, 62 ± 8 and in PR,

n=8, 61 ± 8) to the pre-exercise level (62 ± 8 bpm, n=16). There was significant (p=0.05) association between the recovery rate of heart rate and the recovery mode. Thus, passive recovery explained enhanced heart rate recovery of the PR group. The next morning, the mean heart rate ($HR_{post20h}$) was 64 ± 8 bpm (n=13) which did not differ significantly from the pre-exercise resting HR. In the AR group, the mean $HR_{post20h}$ was 62 ± 9 bmp whereas in the PR group it was 67± 7 bmp, but differed not significantly from the AR groups' value.

8.2.2 Work economy

During the combined loading the mean VO_2 increased significantly ($p < 0.001$) from 15.04 ± 2.03 ml/kg/min to the level of 27.62 ± 3.61 ml/kg/min (n=16). In the AR (n=8) group ($p < 0.001$) mean VO_2 increased from 15.07 ± 1.89 ml/kg/min to 25.43 ± 2.47 ml/kg/min and in the PR (n=8) group ($p < 0.01$) from 15.01 ± 2.30 ml/kg/min to 27.27 ± 5.81 ml/kg/min. (Figure 31.)

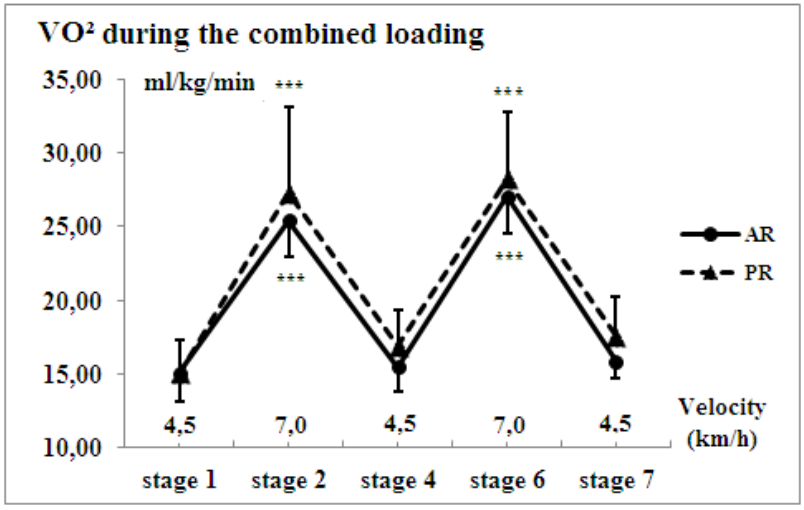

FIGURE 31. The mean oxygen consumption (± SD) of the active (AR) and passive (PR) recovery groups during the combined loading. Significantly different (***= $p < 0.001$) from the stage 1 value.

The mean (n=16) oxygen consumption stayed up (27.03 ± 2.51 mmol/l, n=6, in the AR and 28.21 ± 4.56 ml/kg/min, n=6, in PR group) until the 6th stage and decreased ($p <$

0.001) thereafter to the level of 16.70 ± 2.21 ml/kg/min. The oxygen consumption decreased in the AR (n=8) group ($p < 0.001$) to the level of 15.86 ± 1.14 ml/kg/min and in the PR (n=8) group ($p < 0.001$) to 17.54 ± 2.74 ml/kg/min. Comparing the oxygen consumption values during the identical 1st and 7th stages (4.5 km/h) the cost of walking increased significantly ($p < 0.01$) by average of 1.7 ± 2.0 ml/kg/min, which is about 11 ± 10 % change from stage 1. During the 6th stage (7.0 km/h) the cost of walking did not change significantly. There were no significant differences between the groups.

8.2.3 Blood lactate

During the loading the mean (n=12) blood lactate level increased significantly ($p < 0.001$) by 72 ± 24% (1.11 ± 0.31 mmol/l to 4.02 ± 1.89 mmol/l). In AR (n=6) blood lactate increased ($p < 0.05$) from 1.04 ± 0.33mmol/l to 3.24 ±1.41 mmol/l and in PR (n=6) ($p < 0.001$) from 1.18 ± 0.30 mmol/l to 4.80 ± 2.09 mmol/l. (Figure 32.)

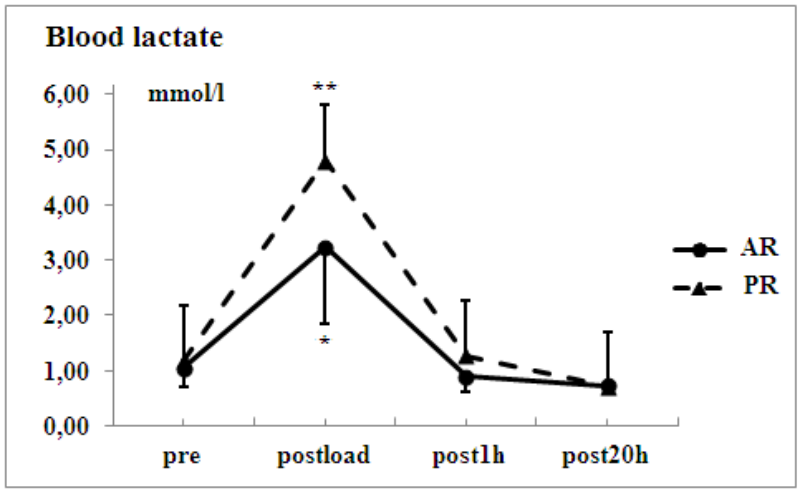

FIGURE 32. The mean (±SD) blood lactate of the active (AR, n=6) and passive (PR, n=6) recovery groups before (pre), immediately after the loading (postload), immediately after the recovery (post1h) and next morning (post20h) in reservists men. Significantly different (** = $p < 0.01$, *= $p < 0.05$) from the pre-exercise value.

However, during the recovery period the mean (n=12) level of blood lactate decreased significantly ($p < 0.001$) back to the pre-exercise level 1.08 ± 0.44 mmol/l (n=12). In the

AR (n=6) group the mean lactate level decreased ($p < 0.05$) to 0.89 ± 0.27 mmol/l and in PR (n=6) group ($p < 0.01$) to 1.26 ± 0.51 mmol/l. The next morning the mean (n=11) lactate level (0.72 ± 0.19 mmol/l) was still decreasing significantly ($p < 0.05$) from the post-recovery value. In AR (n=5) to 0.73 ± 0.15 mmol/l and in PR (n=6) ($p < 0.05$) to 0.71 ± 0.24 mmol/l. The lactate leves did not differ significantly between the AR and PR groups at any time point.

8.3 The relation between subjects' fatigue and recovery rates and their fitness level

There were no correlations between the force or EMG losses or recovery of the loading day and subjects' individual strength level (1-RM) measured in the pre-protocol control-day. The correlation occurred neither in the rise or recovery of the heart rate or blood lactate and strength level (1-RM), whereas, between the strength loss and VO_{2max} a positive correlation (r=-0.49, $p < 0.05$) was identified (figure 33).

The higher the VO_{2max} of the subject in the pre-protocol control measurements, the lower the decrease of his maximal isometric force during combined strength and endurance loading was. Similarly, VO_{2max} was identified to explain 55.9 % ($p < 0.001$) of subjects force decrease and 82.5 % ($p < 0.001$) of the recovery rate of heart rate. In addition, the age of the subjects showed negative (r=-0.51) and significant ($p < 0.05$) connection to recovery rate of heart rate.

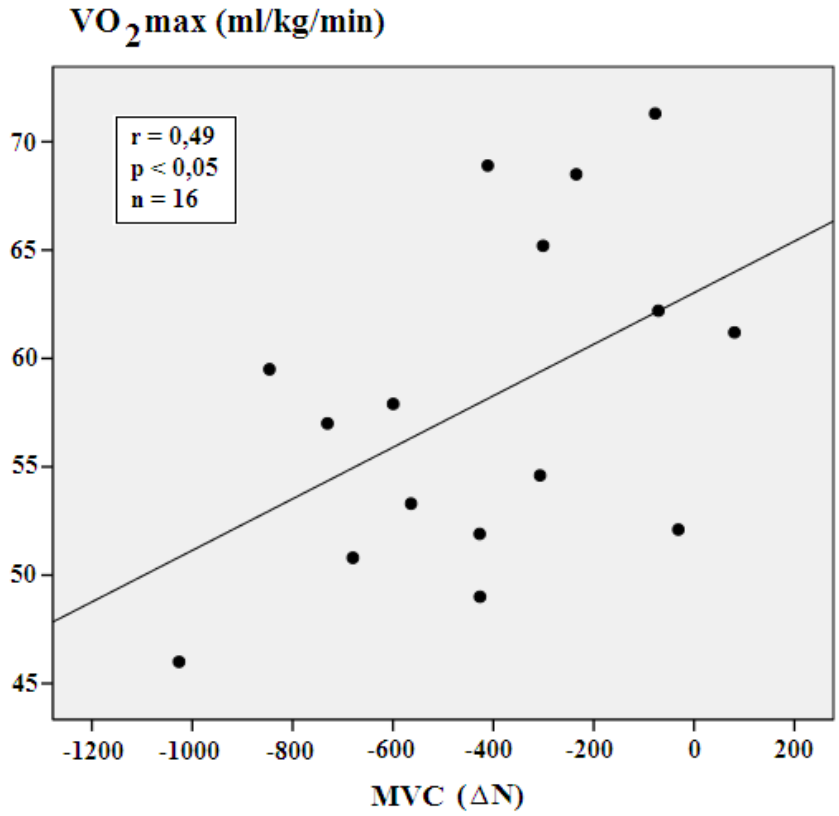

FIGURE 33. The correlation (r^2=0,24) between the combined loading induced strength loss (in MVC) and pre-protocol control day measured VO_{2max} in reservists men (n=16).

9 DISCUSSION

9.1 Primary findings

The purpose of the present study was to examine combined strength and endurance exercise induced acute neuromuscular fatigue and recovery in male reservists. In addition, this study investigated the rate of recovery comparing the active and passive recovery models and the association of the initial fitness levels of subjects on the magnitude of fatigue and recovery rates.

The primary findings of the study were that combined strength and endurance exercise induced significant ($p < 0.001$) decrease in neuromuscular performance measured by maximal isometric force production (MVC), muscle activation (AEMG and iEMG) and rate of force development (force-time curve). In addition, work economy decreased significantly ($p < 0.001$) during the combined loading. However, neuromuscular performance recovered totally during the following 20 hours after the loading. Results of the current study also showed that the active recovery could slightly, but not statistically significantly, enhance the recovery of the subjects' rate of force development.

Interestingly, there was a connection ($p < 0.05$) detected between subjects' pre-protocol fitness level (VO_{2max}) and strength loss. Higher the VO_{2max} in the pre-protocol control measurements lower was the decrease of isometric force during combined strength and endurance loading. Similarly, the higher VO_{2max} of the subjects' was identified ($p < 0.05$) to explain the faster recovery of their heart rate.

9.2 Alteration in neuromuscular performance

Previous studies (Häkkinen 1993, Häkkinen 1994, Häkkinen & Pakarinen 1995, Ahtiainen et al. 2003) have shown that the force generating capacity of muscles becomes progressively impaired during strenuous hypertrophic and heavy resistance exercises and gradually recovers once exercise is terminated. This study found a 14 ± 9% decrease in MVC which was accompanied by the decrease in EMG and downward shift in the force-time curve. However, previous studies have shown hypertrophic type loadings induced alterations in MVC to be much greater than in the present study. In the study of Häkkinen (1994), 10 times 10 repetitions maximum in squat (about 70 % of 1RM) with 3 minutes recovery between sets, induced approximately a 47 % decrease in MVC and in another study of Häkkinen (1993) 20*1RM heavy resistance exercise decreased MVC by 24 %. Therefore, the term "exhaustion" here does not imply complete physical collapse but simply the inability to maintain the intensity or strength level. In addition, in this study regarding combined exercise the decrease in the strength production capacity might be mainly due to strength parts of the combined loading, whereas, the aerobic parts on the treadmill might been actually lightening the stresses of the body.

The decrease in the production of strength has also been attributed to the decrease in neural activation (Komi & Tesch 1979, Häkkinen & Komi 1986). In the present study both the integrated EMG and the averaged EMG declined with declining maximum force generation during combined loading induced acute fatigue. In addition, the rise of blood lactate was identified during the loading. Therefore the present study showed that significant reduction in neuromuscular capacity can be attributed to many factors, as central and peripheral mechanisms of fatigue. However, the decrease in maximal isometric force and right and downward shift in the force-time curve may be due to the decrease in ability to activate motor units (especially type II needed in fast force production) by the central nervous system. The decrease in EMG also supports this suggestion. In addition, part of fatigue response might be also due to accumulation of lactic acid to working muscles which is often associated with the failure to maintain a

given level of muscle force under conditions of exercise-induced fatigue (Mainwood & Renaud 1985, Sahlin et al. 1978).

9.3 Changes in physiological parameters

A few parameters are frequently used to monitor cardiorespiratory, metabolic, and muscular responses to exercise (Nummela et al. 2008b). For example, heart rate is used to monitor exercise intensity, which did not increase too high level in this study (120 ± 25 bpm, n=10). The results of the present study showed that strength parts (stage3, stage5, stage8) and running parts (stage2, stage7) on the treadmill always increased the heart rate level, whereas walking parts (stage1, stage4, stage7) on the treadmill decreased it.

Elevated blood lactate levels observed in the present study have been evident after intensive muscular work also in other studies regarding combined exercise (Leveritt et al. 1999, Leveritt et al. 2000, Brunetti et al. 2008, Holviala et al. 2010) and hypertrophic and heavy resistance exercises (Linnamo et al. 2000, Ahtiainen et al. 2003). For example, in a study of Linnamo et al. (2000) leg press exercise (5*10*70%) elevated lactate concentration to little higher (4.95 ± 0.81 mmol/l) than in this study (4.02 ± 1.89 mmol/l, n=12). Anyway, blood lactate is widely used to determine energy production from glycolytic metabolism. In addition, the rate of perceived exertion (RPE) has been shown to be related to HR and blood lactate during sub-maximal exercise, allowing estimation of exercise intensity using a subjective scale (Desgorces et al. 2007).

Also the oxygen consumption required at a given intensity of exercise is widely used to determine exercise-induced fatigue and was employed in this study. The aerobic parts on the treadmill showed that combined loading resulted in an increase in oxygen consumption and ventilation, and in the decrease (11%) in work economy (VO_2 /km/h) of the subjects. The change of work economy could be explained by several mechanisms: increased utilization of fat as an energy substrate (Ament &Verkerke

2009), increased demands of body temperature regulation, and possible muscle damage (Marcora & Bosio 2007). However, the study of Kyröläinen et al. (2000) showed clearly that weakened running economy cannot be explained by changes in running mechanics (Morgan 1990). Therefore, it is suggested that the increased physiological loading due to combined strength and endurance loading with external load affects negatively to the subjects' running/walking capacity. Possible mechanisms behind the fatigue include a higher central motor command necessary to produce the same running speed with weaker leg muscles, the contribution of leg muscle pain to overall RPE, and the alterations in glycogen metabolism and availability (Kyröläinen et al. 2000).

9.4 Active and passive recovery

Previous studies considering hypertrophic and heavy resistance exercises have shown how isometric strength is reduced after intensive muscular work and recovery takes from hours to several days (Häkkinen 1993, Häkkinen1994, Häkkinen 1995, Linnamo et al. 2000). Therefore, the aim of the present study was to examine the effects of active and passive recovery on performance. Generally, previous studies have shown passive recovery to reduce the resting metabolic needs (Harper et al. 2008) and active recovery facilitated the strength recovery and lactate removal (Bogdanis et al. 1996).

Several studies have shown that the recovery of maximal force is fast but not complete during the first hour of rest (Häkkinen 1993, Linnamo 1993, 68, Häkkinen 1994, Häkkinen 1995) and this study does not make an exception. One explanation for the fast recovery of force might be the simultaneous decrease in blood lactate which was also identified in this study, whereas, the relationship between the different recovery modalities and the subjects' force generation capacity is much more unclear (Corder et al. 2000). In the present study, maximal isometric force and rate of force development recovered following 20h the combined loading, and there were no statistically significant differences in the recovery rate between the active and passive recovery groups. However, the force-time curve of the active recovery group recovered clearly,

but not statistically significantly, during the 1h recovery period when the force-time curve of the passive recovery group stayed decreased. Also the absolute level of maximal isometric force (measured in the next morning) increased in the active recovery group slightly (36 ± 322 N, n=8) above the pre-exercise value whereas the force level of the passive recovery group stayed (18 ± 243 N, n=8) below their pre-exercise value. The finding is also in line with Lattier et al. (2004) who observed the tendency for better force generation after active recovery.

Chrisafulli et al. (2003) studied the differences in cardiovascular response during two modes of recovery. Not surprisingly, the heart rate recovery also in this present study was significantly related to the recovery model being faster in the passive recovery group, which is in line with Chrisafulli et al. (2003). Thus, the difference is fully explained by the higher muscular engagement involved in active recovery compared to passive recovery. However, 20h after the combined loading the mean heart rate was clearly, but not statistically significantly, lower in the active recovery group (62 ± 9 bmp, n=8) compared to the passive recovery group (67± 7 bmp, n=8), althought before the loading the levels of the groups were almost the same (in AR, n=8, 63 ± 8 bpm and in PR, n=8, 62 ± 7 bmp). Because the HR value also reflects the overall rate of blood flow, which in turn illustrates the state of blood lactate removal, using HR as a cardiovascular marker followed by different recovery interventions can give a proper picture of the overall recovery state (Barnett 2006).

The latter, blood lacate removal, is also closely associated with muscle fatigue. Several studies comparing the active and passive way to recover have reported lower lactate concentrations following active recovery (Coffey et al. 2004, Greenwood et al. 2008, Bangsbo et al. 1994) but in this study the lactate levels recovered to the pre-exercise value in both groups during the 1h recovery period. In addition, the lactate removal was actually faster in the passive recovery group because their absolute level (4.80 ± 2.09 mmol/l, n=6) of blood lactate was higher than in the active recovery groups' (3.24 ±1.41 mmol/l, n=6) immediately after the loading. Anyway, the finding has been observed also in other studies. For example, even if the performance had enhanced after active

recovery (Bogdanis et al. 1996) the blood lactate and pH were not changed by active recovery (Toubekis et al. 2008).

All in all, the process of recovery taking place during rest after the combined strength and endurance exercise loading need further examination. The study showed some tendencies that the active recovery mode has advantages in terms of recovery of fast force production. Passive recovery instead seemed to remove the blood lactate more effectively than the active recovery. Whereas, active recovery slowered the heart rate recovery in the loading day but decreased the resting heart rate level in the next morning. Therefore, althought the active recovery is recommended to increase performance (Thevenet et al. 2008), more specific examination is required to enhance the rate of recovery in order to avoid overstress and to improve the overall training.

9.5 Other factors affecting the rates of fatigue and recovery

While the exact causes of muscle fatigue and the relative importance of particular factors remain complicated, it is suggested that an individual's state of fitness, dietary status, fiber type composition, and intensity and duration of the exercise all affect the process. The magnitude of the decrease in muscular strength during fatigue may be related to the type of fatiguing load (Komi & Viitasalo 1977), to the recovery time between sets (Komi & Tesch 1979) and to the subject material. In this study, the overall load did not increase very high because of the design of the study. The times between the different stages (strength vs endurance) which consisted of changing the cloths (taking off and on the weighted vest, military boots, helmet and the riffle) and moving from one place to another (between the strength laboratory and treadmill) gave subjects some time to recover and affected the overall loading.

However, the fatigue response of a muscle is a complex process which involves a number of factors in the muscle contractile machine, the nervous system and the

metabolic responses (Kent-Braum 1999). The problem is further complicated as muscle fatigue is not only a result from multiple factors acting at various sites but in many cases from synergistic actions of two or more agents (Fitts 2008). For example, the subjects material was a bit different (significant difference in body mass) between the active and passive recovery groups, which was identified in higher (but not significantly differed) force levels and lower heart rate and blood lactate levels in the active recovery group. On the contrary, the dietary status of the subjects was regulated by 12h fasting before the combined strength and endurance exercise.

Interestingly, the study found a negative correlation between the individuals' VO_{2max} and strength loss, which indicates the importance of individual fitness state in acute fatigue. The higher the VO_{2max} of the subject in the pre-protocol control measurements, the lesser the decrease of his maximal isometric force during combined strength and endurance loading was. Parallel findings were made by Häkkinen & Myllylä (1990) who identified maintained 60% isometric loading induced worsening in maximal force as well as in the maximal rates of force production being significantly smaller in endurance athletes than the corresponding decreases in power or strength athletes. Among the others, Behm & St-Pierre (1997) have explained the phenomenon by the finding that muscles with higher percentages of fast-twitch fibers have been shown to fatigue more rapidly than do muscles with a greater percentage of slow-twitch fibers.

According to Tesch et al. (1986), during a hypertrophic type strength loading, the muscle glycogen decreases 40% and is regenerated during the recovery. Usually, the level of glycogen recovers to the pre-exercise quantity during following 24h, if the carbohydrate supply is adequate and the muscle damage not too wide (Trappe et al. 2002, Bogdanis et al. 1996). Therefore, it can be concluded that the next exercise session could be done after 20-24 hours recovery which is supported by the results of the present study. In addition, restoration of the body's water balance is an important part of the recovery process after exercise and can be achieved by ingestion of water if consumed in sufficient volume together with a meal providing significant amounts of electrolytes (Maughan et al. 1996).

When exercise is terminated the recovery of fatigued muscles starts immediately and is also related to the training status and to the individual characreristics. According to Tesch & Wright (1983), individuals who are easily fatigued recover slower than the persons in better physical condition. The results of the present study also pointed out the advantage of the better fitness in terms of heart rate recovery. Maximal oxygen consumption was identified to explain 82.5 % about subjects' heart rate recovery which signified that the better the endurance capacity, the faster the recovery of heart rate of the subject was. Several studies before have shown how maximal oxygen uptake is most often the reason for limitations to endurance capacity of prolonged load carrying performance (Holviala et al. 2010) but this study indicated that the endurance capacity of the subject is related to the responses of fatigue but also the recovery.

Surprisingly, there were no correlations identified between the fatigue or recovery responses and 1-RM of the subjects. In conclusion, although there is no existing comprehensive explanation about the fatigue, according to the results of the present study the endurance capacity of the subject affects both the fatigue and recovery more than the subject's strength capacity does.

10 CONCLUSIONS

The main aim of this study was to determine if and for how long strength and muscle activation are reduced after an acute bout of combined strength and endurance exercise. The findings of the present study indicated that strenuous combined loading can result in acute fatigue, which can be observed as decrease in force production characteristics and muscle activation of loaded muscles. The findings of the current study also showed that the loaded treadmill walking affects walking efficiency negatively.

These results of the study suggest that individuals performing combined strength and endurance loading are able to perform the next exercise session 20h after combined exercise without any significant reduction in force production. The endurance capacity of the subject also affected the rate of fatigue and recovery, whereas the strength capacity did not show any effect.

The study also examined the advantages of active recovery and some tendencies in terms of enhancing recovery of fast force production were identified. Therefore, it can be revealed that light aerobic activity could be chosen after strenuous exercise for enhancing recovery, which is widely used in many sports disciplines, and could also be beneficial in military settings.

11 REFERENCES

ACSM's guidelines for exercise testing and prescription. 2006. Philadelphia: Lippincott Williams & Wilkins.

Adlercreutz, H., Härkönen, M., Kuoppasalmi, K., Näveri, H., Huhtaniemi, I., Tikkanen, H., Remes, K., Dessypris, A. & Karvonen, J. 1986. Effect of training on plasma anabolic and catabolic steroid hormones and their response during physical exercise. International Journal of Sports Medicine 7, 27-28.

Ahtiainen, J. P., Pakarinen, A., Kraemer, W. J & Häkkinen, K. 2003. Acute hormonal and neuromuscular responses and recovery to forced vs. maximum repetitions multiple resistance exercises. International Journal of Sports Medicine 24, 410-418.

Ahtiainen, J. P., Pakarinen, A., Alen M., Kraemer, W. J. & Häkkinen, K. 2005. Short vs. long rest period between the sets in hypertrophic resistance training. Influence on muscle strength, size, and hormonal adaptations in trained men. Journal of Strength and Conditioning Research 19, 3, 572-582.

Ahtiainen, J. P., Hulmi, J. J., Kraemer, W. J., Lehti, M., Pakarinen, A., Mero, A. A., Karavirta, L., Sillanpää, E., Selänne, H., Alen, M., Komulainen, J., Kovanen, V., Nyman, K. & Häkkinen, K. 2009. Strength, endurance or combined training elicit diverse skeletal muscle myosin heavy chain isoform proportion but unaltered androgen receptor concentration in older men. International Journal of Sports Medicine 30, 12, 879-887.

Ament, W. & Verkerke, G. J. 2009. Exercise and fatigue. Sports Medicine 39, 389-422.

Avela, J., Kyröläinen, H. & Komi, P. V. 2001. Neuromuscular changes after long-lasting mechanically and electrically elicited fatigue. European Journal of Applied Physiology 85, 317-125.

Baldari, C., Di Luigi, L., Da Silva, S. G., Gallotta, M. C., Emerenziani, G. P., Pesce, C. & Guidetti, L. 2007. Relationship between optimal lactate removal power output

and olympic triathlon performance. Journal of Strength and Conditioning Research 21, 1160-1165.

Bangsbo, J., Graham, T., Johansen, L. & Saltin, B. 1994. Muscle lactate metabolism in recovery from intense exhaustive exercise: impact of light exercise. Journal of Applied Physiology 77, 1890-1895.

Barnett, A. 2006. Using recovery modalities between training sessions in elite athletes: Does it help? Sports Medicine 36, 9, 781-796.

Behm, D. G. & St-Pierre, D. M. M. 1997. Effects of fatigue duration and muscle type on voluntary and evoked contractile properties. Journal of Applied Physiology 82, 1654-1661.

Bell, G. J., Syrotuik, D. Martin, T. P., Bumham, R. & Quinney, H. A. 2000.The effect of concurrent strength and endurance training on skeletal muscle properties and hormone concentrations. European Journal of Applied Physiology 81, 418-427.

Bigland-Ritchie, B. & Woods, J. J. 1984 Changes in muscle contractile properties and neural control during human muscular fatigue. Muscle Nerve. 7, 9, 691-699.

Bogdanis, G. C., Nevill, M. E., Lakomy, H. K. A., Graham, C. M. & Louis, G. 1996. Effects of active recovery on power output during repeated maximal sprint cycling. European Journal of Applied Physiology 74, 461-469.

Borg, G.A. 1982. Psychophysical bases of perceived exertion. Medicine and Science in Sports and Exercise 14, 5, 377-381.

Brooks, G. A. & Fahey, T. D. 1984. Exercise Physiology: Human Bioenergetics and Its Applications. Canada: John Wiley & Sons.

Brunetti, A. P., Adolfo, J., Brum, P. P., Sampaio, V. M., Dantas, E. H. M. & dos Santos, M. A. A. 2008. Training session over the acute response of the blood lactate, heart rate and oxygen consumption. Fittnes & Performance Journal 7, 326-331.

Bulbulian, R., Heaney, J. H., Leake, C. N., Sucec, A. A. & Sjoholm, N. T. 1996. The effect of sleep deprivation and exercise load on isokinetic leg strength and endurance. European Journal of Applied Physiology 73, 273-277.

Byrne, C. & Eston, R. 2002. The effect of exercise-induced muscle damage on isometric and dynamic knee extensor strength and vertical jump performance. Journal of Sports Sciences, 20, 417- 425.

Byrne, C., Eston, R. G. & Edwards, H. T. 2001. Characteristics of isometric and dynamic strength loss following eccentric exercise-induced muscle damage. Scandinavian Journal of Medicine & Science in Sports 11, 134-140.

Cairns, S. P. 2006. Lactic acid and exercise performance: Culprit or friend? Sports Medicine 36, 4, 279-291.

Calbet, J. A. L., Rådegran, G., Boushel, R. & Saltin, B. 2009. On the mechanisms that limit oxygen uptake during exercise in acute and chronic hypoxia: role of muscle mass. The Journal of Physiology 587, 2, 477–490.

Castagna, C., Abt, G., Manzi, V., Annino, G., Padua, E. & D'Ottavio, S. 2008. Effects of recovery mode on repeated sprint ability in young basketball players. Journal of Strength and Conditioning Research 22, 923–929.

Chilibeck, P. D., Syrotuik, D. G. & Bell, G. J. 2002. The effect of concurrent endurance and strength training on quantitative estimates of subsarcolemmal and intermyofibrillar mitochondria. International Journal of Sports Medicine 23, 33-39.

Chrisafulli, A., Orrù, V., Melis, F., Tocco, F. & Concu, A. 2003. Hemodynamics during active and passive recovery from a single bout of supramaximal exercise. European Journal of Applied Physiology 89, 2, 209-216.

Christmass, M. A., Dawson, B. & Arthur, P. G. 1999. Effect of work and recovery duration on skeletal muscle oxygenation. European Journal of Applied Physiology 80, 436-447.

Chtara, M., Chaouachi, A., Levin, G. T., Chaouachi, M., Chamari, K., Amri, M. & Laursen, P. B. 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.

Coffey,V., Leveritt, M. & Gill, N. 2004. Effect of recovery modality on 4hour repeated treadmill running performance and changes in physiological variables. Journal of Science and Medicine in Sport 7, 1, 1-10.

Cooke, M. B., Rybalka, E., Williams, A. D., Cribb, P. J. & Hayes, A. 2009. Creatine supplementation enhances muscle force recovery after eccentrically-induced muscle damage in healthy individuals. Journal of the International Society of Sports Nutrition 6, 13, 1-11.

Corder, K. P., Potteiger, J. A., Nau, K. L., Figoni, S. E. & Hershberger, S. L. 2000. Effects of active and passive recovery conditions on blood lactate, rating of perceived exertion, and performance during resistance exercise. Journal of Strength & Conditioning Research 14, 2, 151-156.

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 and Exercise Science 5, 281-285.

Desgorces, F. D., Senegas, X., Garcia, J., Decker, L. & Noirez, P. 2007. Methods to quantify intermittent exercises. Applied Physiology, Nutrition and Metabolism 32, 762–769.

Doherty, D. & Sporer, B. 2000. A proposed model for examining the interference phenomenon between concurrent aerobic and strength training. Sports Medicine 30, 6, 385-394.

Dousset, E., Avela, J., Ishikawa, M., Kallio, J., Kuitunen, S., Kyröläinen, H., Linnamo, V. & Komi, P. V. 2007. Bimodal recovery pattern in human skeletal muscle induced by exhaustive stretch-shortening cycle exercise. Medicine & Science in Sports & Exercise 39, 3, 453-460.

Duffield, R., King, M. & Skein, M. 2009. Recovery of voluntary and evoked muscle performance following intermittent-sprint exercise in the heat. International Journal of Sports Physiology and Performance 4, 254-268.

Enoka, R. M. 2002. Neuromechanics of Human Movement. Champaign (IL): Human Kinetics.

Ferketich, A. K., Kirby, T. E. & Alway, S. E. 1998. Cardiovascular and muscular adaptations to combined endurance and strength training in elderly women. Acta Physiologica Scandinavica 164, 259-267.

Fitts, R. H. 2008. The cross-bridge cycle and skeletal muscle fatigue. Journal of Applied Physiology 104, 551–558.

Fitts, R. H. & Holloszy, J. O. 1976. Lactate and contractile force in frog muscle during development of fatigue and recovery. American Journal of Physiology 231, 430-433.

Greenwood, J.D., Moses, G. E., Bernardino, F. M., Gaesser, G. A. & Weltman, A. 2008. Intensity of exercise recovery, blood lactate disappearance, and subsequent swimming performance. Journal of Sports Sciences 26, 29-34.

Harper, A. J., Ferreira, L. F., Lutjemeier, B. J., Townsend, D. K. & Barstow, T. J. 2008. Matching blood flow to metabolic rate during recovery from moderate exercise in humans. Experimental Physiology 93, 10, 1118-1125.

Hawley, J. A. 2009. Molecular responses to strength and endurance training: are they incompatible? Applied Physiology, Nutrition and Metabolism 34, 355-361.

Hickson, R. C. 1980. Interference of strength development by simultaneously training for strength and endurance. European Journal of Applied Physiology and Occupational Physiology 45, 255-263.

Holviala, J., Häkkinen, A., Karavirta, L., Nyman, K., izquierdo, M., Gorostiaga, E. M., Avela, J., Korhonen, J., Knuutila, V-P., Kraemer, W. J. & Häkkinen, K. 2010. Effects of combined strength and endurance training on treadmill load carrying walking performance in aging men. Journal of Strength and Conditioning Research 24, 6, 1584-1595.

Horita, T., Komi, P.V., Nicol, C. & Kyröläinen, H. 1999. Effect of exhausting stretch-shortening cycle exercise on the time course of mechanical behaviour in the drop jump: possible role of muscle damage. European Journal of Applied Physiology 79, 160-167.

Häkkinen, K. & Komi, P. 1986. Effects of fatigue and recovery on electromyographic and isometric force and relaxation-time characteristics of human skeletal muscle. European Journal of Applied Physiology 55, 588-596.

Häkkinen, K., Kauhanen, H. & Komi, P. V. 1988a. Effects of fatiguing loading with a variable resistance equipment on neural activation and force production of the knee extensor muscles. Electromyography and Clinical Neurophysiology 28, 79-87.

Häkkinen, K., Pakarinen, A., Alén, M., Kauhanen, H. & Komi, P.V. 1988b. Neuromuscular and hormonal responses in elite athletes to two successive strength training sessions in one day. European Journal of Applied Physiology and Occupational Physiology 57, 2, 133-139.

Häkkinen, K, & Myllylä, E.1990. Acute effects of muscle fatigue and recovery on force production and relaxation in endurance, power and strength athletes. The Journal of Sports Medicine and Physical Fitness 1, 5-12.

Häkkinen, K. 1992. Neuromuscular responses in male and female athletes to two successive strength training sessions in one day. The Journal of Sports Medicine and Physical Fitness 32, 3, 234-242.

Häkkinen K. 1993. Neuromuscular fatigue and recovery in male and female athletes during heavy resistance exercise. International Journal of Sports Medicine 14, 2, 53-59.

Häkkinen, K. & Pakarinen, A. 1993. Acute hormonal responses to two different fatiguing heavy-resistance protocols in male athletes. Journal of Applied Physiology 74, 882–887.

Häkkinen, K. 1994. Neuromuscular fatigue in males and females during strenuous heavy resistance loading. Electromyography and Clinical Neurophysiolology 34, 4, 205-214.

Häkkinen K.1995. Neuromuscular fatigue and recovery in women at different ages during heavy resistance loading. Electromyography and Clinical Neurophysiolology 35, 7, 403-413.

Häkkinen, K. & Pakarinen, A. 1995. Acute hormonal response to heavy resistance loading in men and women at different ages. International Journal of Sports Medicine 16, 507-513.

Häkkinen, K., Alen, M., Kraemer, W. J., Gorostiaga, E., Izquierdo, M., Rusko, H., Mikkola, J., Häkkinen, A.,Valkeinen, H., Kaarakainen, E., Romu, S., Erola, V., Ahtiainen, J. & L. Paavolainen. 2003. Neuromuscular adaptations during concurrent strength and endurance training versus strength training. European Journal of Applied Physiology 89, 42–52.

Ivy, J. L., Katz, A. L., Culter, C. L., Sherman, W. M. & Coyle, E. F. 1988. Muscle glycogen synthesis after exercise: Effect of time of carbohydrate ingestion. Journal of Applied Physiology 64, 1480-1485.

Izquierdo, M., Häkkinen, K., Ibanez, J., Kraemer, W. J. & Gorostiaga, E. M. 2005. Effects of combined resistance and cardiovascular training on strength, power, muscle cross-sectional area, and endurance markers in middle-aged men. European Journal of Applied Physiology 94, 70-75.

Izquierdo, M., Ibanez, J., Calbet, J. A. L., Gonzalez-Izal, M., Navarro-Amezqueta, I., Granados, C., Malanda, A., Idoate, F., Gonzalez-Badillo, J. J., Häkkinen, K., Kraemer, W. J., Tirapu, I. & Gorostiaga, E. M. 2009. Neuromuscular fatigue after resistance training. International Journal of Sports Medicine 30, 614-623.

Karatzaferi, C., Giakas, G. & Ball, D. 1999. Fatigue profile: a numerical method to examine fatigue in cycle ergometry. European Journal of Applied Physiology 80, 508-510.

Kawabata, Y., Senda, M., Oka, T., Yagata, Y., Takahara, Y., Nagashima, H. & Inoue, H. 2000. Measurement of fatigue in knee flexor and extensor muscles. Acta Medica Okayama 54, 85-90.

Kent-Braum, 1999. Central and peripheral contributions to muscvle fatigue in humans during sustained maximal effort. European Journal of Applied Physiology 80, 57-63.

Komi, P. V. & Bosco, C. 1978. Utilization of stored elastic energy in leg extensor muscles by men and women. Medicine and Science in Sports and Exercise 10, 261-265.

Komi, P. & Tesch, P. 1979. EMG frequency spectrum, muscle structure and fatigue during dynamic contractions in man. European Journal of Applied Physiology 42, 41-50.

Komi, P. V. & Nicol, C. 2000. Stretch-shortening cycle of muscle function. In: Biomechanics in Sport (eds. V. M. Zatsiorsky). Blackwell Science 2000.

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, 976-989.

Kraemer, W.J. & Ratamess N.A. 2005. Hormonal responses and adaptations to resistance exercise and training. Sports Medicine 35, 4, 339-361.

Kubo, K., Kanehisa, H. & Fukunaga, T. 2005. Influences of repetitive drop jump and isometric leg press exercises on tendon properties in knee extensors. Journal of Strength and Conditioning Research 19, 864-870.

Kuoppasalmi 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 and Laboratory Investigation 40, 5, 403-9.

Kyröläinen, H., Häkkinen, K., Kautiainen, H., Santtila, M., Pihlainen, K. & Häkkinen, A. 2008. Physical fitness, BMI and sickness absence in male military personnel. Occupational Medicine 58, 4, 251 - 256.

Kyröläinen, H., Pullinen, T., Candau, R., Avela, J., Huttunen, P. and Komi, P. V. 2000. Effects of marathon running on running economy and kinematics. European Journal of Applied Physiology 82, 4, 297-304.

Lattier, G., Millet, G.Y., Martin, A. & Martin, V. 2004. Fatigue and recovery after high-intensity exercise. Part II: Recovery interventions. International Journal of Sports Medicine 25, 509-515.

Leveritt, M., Abernethy, P. J., Barry, B. K. & Logan, P. A. 1999. Concurrent strength and endurance training. Sports Medicine 28, 413-427.

Leveritt, M. & Abernethy, P. J. 1999. Effects of carbohydrate restriction on strength performance. Journal of Strength & Conditioning Research 13, 1, 52-57.

Leveritt, M., MacLaughlin, H. & Abernethy, P. J. 2000. Changes in leg strength 8 and 32 h after endurance exercise. Journal of Sports Sciences 18, 865 – 871.

Linnamo, V. 1993. Maksimivoima- ja nopeusvoimaharjoituksen akuutit vaikutukset neuromuskulaariseen väsymykseen ja palautumiseen. Jyväskylä University. Master Thesis of Biomechanics.

Linnamo, V., Newton, R. U., Häkkinen, K., Komi, P. V., Davie, A., McGuigan, M. & Triplett-McBride. 2000. Neuromuscular responses to explosive and heavy resistance loading. Journal of Electromyography and Kinesiology 10, 6, 417-424.

Linnamo, V., Pakarinen, A., Komi, P.V., Kraemer, W.J. and Häkkinen. K. 2005. Acute hormonal responses to submaximal and maximal heavy resistance and explosive exercises in men and women. J. Strength Cond. Res. 19, 3, 566–571.

Mainwood, G. W., & Renaud, J. M. 1985. The effect of acid-base balance on fatigue of skeletal muscle. Canadian Journal of Physiology and Pharmacology 63, 5, 403-16.

Mannion, A. F. & Dolan, P. 1996. Relationship between myoelectric and mechanical manifestations of fatigue in the quadriceps femoris muscle group. European Journal of Applied Physiology 74, 411-419.

Marcora, S. M. & Bosio, A. 2007. Effect of exercise-induced muscle damage on endurance running performance in humans. Scandinavian Journal of Medicine and Science in Sports 17, 662-671.

Maughan, R. J., Leiper, J. B. & Shirreffs. 1996. Restoration of fluid balance after exercise-induced dehydration: effects of food and fluid intake. European Journal of Applied Physiology 73, 317-325.

Mikkola, J. S., Rusko, H. K., Nummela, A., Paavolainen, L. M. & Häkkinen, K. 2007. Concurrent endurance and explosive type strength training increase activation and fast force production of leg extensor muscles in endurance athletes. Journal of Strength and Conditioning Research 21, 613-620.

Millet, G.Y. & Lepers, R. 2004. Alterations of neuromuscular function after prolonged running, cycling and skiing exercises. Sports Medicine 34, 2, 105-116.

Morgan, D. L. 1990. New insights into the behavior of muscle during active lengthening. Biophysics Journal 57, 209–221.

Nader, G. A. 2006. Concurrent strength and endurance training: from molecules to man. Medicine and Science in Sports & Exercise 11, 1965-1970.

Nicol, C., Komi, P. V., Horita, T., Kyröläinen, H. & Takala, T. E. S. 1996. Reduced stretch-reflex sensitivity after exhausting stretch-shortening cycle exercise. European Journal of Applied Physiology 72, 401-409.

Nindl, B. C., Leone, C. D., Tharion, W., Johnson, R.F., Castellani, J., Patton, J. F. & Montain, S. J. 2002. Physical performance responses during 72 h of military operational stress. Medicine & Science in Sports & Exercise 34, 11, 1814-1822.

Nummela, A. T., Heath, K. A., Paavolainen, L. M., Lambert, M. I. St Clair Gibson, A., Rusko, H. K. & Noakes, T. D. 2008a. Fatigue during a 5-km Running Time Trial. International Journal of Sports Medicine 29, 738-745.

Nummela, A. T., Kaikkonen, P. J. & Rusko, H. K. 2008b. Methods for determining training load of submaximal running exercises. Medicine & Science in Sports & Exercise 40, 5, 41.

Osborne, M. A. & Schneider, D. A. 2006. Muscle glycogen reduction in man: relationship between surface EMG activity and oxygen uptake kinetics during heavy exercise. Experimental Physiology 91, 1, 179-189.

Paavolainen, L., Häkkinen, K., Nummela, A. & Rusko, H. 1994. Neuromuscular characteristics and fatigue in endurance and sprint athletes during a new anaerobic power test. European Journal of Applied Physiology 69, 119-126.

Paavolainen, L. M., Häkkinen, K., Nummela, A. T. & Rusko, H. K. 1997. Neuromuscular characteristics and fatigue as determinants of distance running performance. Medicine & Science in Sports & Exercise 29, 5, 280.

Paavolainen, L., Häkkinen, K., Hämäläinen, I., Nummela, A. & Rusko, H. 1999. Explosive-strength training improves 5-km running time by improving running economy and muscle power. Journal of Applied Physiology 86, 5, 1527-1533.

Piitulainen, H., Kivelä, R., Komi, P., Kainulainen, H. & Kyröläinen, H. 2008. Molecular adaptations of voltage-gated sodium ion channel related proteins after fatiguing stretch-shortening cycle exercise. Scandinavian Journal of Medicine & Science in Sports 18, 636-642.

Price, M. & Moss, P. 2007. The effects of work: rest duration on physiological and perceptual responses during intermittent exercise and performance. Journal of Sports Sciences 25, 14, 1613 – 1621.

Pullinen, T., Leynaert, M. & Komi, P. V. 1997. Neuromuscular function after marathon. Abstract Book of the XVI ISB Congress, August 24-27. Tokyo.

Sahlin, K., Tonkonogi, M. & Söderlund, K. 1999. Plasma hypoxanthine and ammonia in humans during prolonged exercise. European Journal of Applied Physiology 80, 417-422.

Sahlin, K., Alvestrand, A., Brandt, R. & Hultman, E. 1978. Intracellular pH and bicarbonate concentration in human muscle during recovery from exercise. Journal of Applied Physiology 45, 3, 474-480.

Schumann, M. 2011. Acute blood lactate and hormonal responses to single session combined strength and endurance loadings: Order effect in female and male endurance runners. Master's Thesis in Science of Sport Coaching and Fitness testing. Department of Biology of Physical Activity. University of Jyväskylä.

Senna, G. Salles, B. F., Prestes, J., Mello, R. A. & Simao, R. 2008. Influence of two different rest interval lengths in resistance training sessions for upper and lower body. Journal of Sports Science and Medicine 8, 197-202.

Sharwood, K., Paavolainen, L., Nummela, A,. Lambert, M,. Gibson, A., St Clair., Rusko, H. & Noakes, T. 2003. Changes in neural recruitment patterns during and after a 5km time trial in distance runners. Medicine & Science in Sports & Exercise 35, 5, 102.

Sidhu, S. K., Bentley, D. J. & Carroll, T. J. 2009. Locomotor exercise induces long lasting impairments in the capacity of the human motor cortex to voluntarily activate knee extensor muscles. Applied Physiology 106, 556–565.

Siegler, J. C., Bell-Wilson, 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 Sport Nutrition and Exercise Metabolism 16, 92-107.

Skof, B. & Strojnik, V. 2005. Neuromuscular fatique and recovery dynamics following prolonged continuous run at anaerobic threshold. British Journal of Sports Medicine 40, 219-222.

de Souza, E. O., Tricoli, V., Franchini, E., Paulo, A. C., Regazzini, M. & Ugrinowitsch, C. 2007. Acute effect of two aerobic exercise modes on maximum strength and strength endurance. Journal of Strength and Conditioning Research 21, 1286-1290.

Sporer, B. C. & Wenger, H.A. 2003. Effects of aerobic exercise on strength performance following various periods of recovery. The Journal of Strength & Conditioning Research 17, 4, 188-192.

Tesch, P. A. & Wright, J. E. 1983. Recovery from short term intense exercise: Its relation to capillary supply and blood lactate concentration. European Journal of Applied Physiology 52, 98-103.

Tesch, P. A, Colliander, E. B. & Kaiser, P. 1986. Muscle metabolism during intense, heavy-resistance exercise. European Journal of Applied Physiology 55, 362-366.

Thevenet, D., Leclair, E., Tardieu-Berger, M., Berthoin, S., Regueme, S. & Prioux, J. 2008. Influence of recovery intensity on time spent at maximal oxygen uptake during an intermittent session in young, endurance-trained athletes. Journal of Sports Sciences 26, 1313–1321.

Toubekis, A. G., Peyrebrune, M. C., Lakomy, H. K. A. & Nevill, M. E. 2008. Effects of active and passive recovery on performance during repeated-sprint swimming. Journal of Sports Sciences 26, 1497–1505.

Tremblay, M. S., Copeland, J. L. & Van Helder, W. 2005. Influence of exercise duration on post-exercise steroid hormone responses in trained males. European Journal of Applied Physiology 94, 5-6, 505-513.

Viitasalo, J. H. T. & Komi, P. V. 1977. Signal characteristics of EMG during fatigue. European Journal of Applied Physiology 37, 111-121.

Viitasalo, J. T., Salo, A. & Lahtinen, J. 1998. Neuromuscular functioning of athletes and non-athletes in the drop jump. European Journal of Applied Physiology, 78, 432–440.

Viru, A. M., Hackney, A.C., Välja, E., Karleson, K., Janson, T. & Viru, M. 2001. Influence of prolonged continuous exercise on hormone responses to subsequent exercise in humans. European Journal of Applied Physiology 85, 578-585.

Vuorimaa. T., Vasankari. T., Mattila. K., Heinonen, O., Häkkinen, K. & Rusko, H. 1999. Serum hormone and myocellular protein recovery after intermittent runs at the velocity associated with VO (2max). Eur J Appl Physiol Occup Physiol. 80, 6, 575-581.

Yeung, S. S., Au, A. L. & Chow, C. C. 1999. Effects of fatigue on the temporal neuromuscular control of vastus medialis muscle in humans. European Journal of Applied Physiology 80, 379-385.

Zainuddin, Z., Hope, P., Newton, M., Sacco P. & Nosaka, K. 2005. Effects of partial immobilization after eccentric exercise on recovery from muscle damage. Journal of Athletic Training , 40, 197–202.

APPENDIX 1 Informed consent form

Minua on pyydetty osallistumaan tutkimukseen **"Reserviläisten fyysinen suorituskyky 2008"**. Vakuutan, että luettuani koehenkilötiedotteen ja saatuani haluamani lisäinformaation suostun tutkimukseen vapaaehtoisesti ja ilman painostusta. Minulla on ollut riittävästi aikaa harkita osallistumistani. En ole lääketieteellisestä tutkimuksesta annetun lain 488/1999 7-10 §:n tarkoittama henkilö (alaikäinen, raskaana/imettävä nainen, vanki tai kehitysvammaisuuden/mielenterveyshäiriön/muun syyn vuoksi vajaakykyinen). Tiedän voivani milloin tahansa peruuttaa suostumukseni ja siten keskeyttää osallistumiseni tutkimukseen sen vaikuttamatta oikeuksiini. Ymmärrän tutkimuksen hyödyt ja haitat. Mikäli haittavaikutuksia ilmenee, lupaan viipymättä ilmoittaa niistä tutkimushenkilökunnalle.

Suostun siihen, että tutkimuksen suorituksen aikana, mikäli se on oman turvallisuuteni kannalta tarpeellista, tutkimusta suorittavat lääkärit saavat hankkia itseäni koskevia tietoja niistä terveydenhuollon yksiköistä, joissa olen aiemmin ollut hoidettavana tai tutkittavana. Tiedän, että henkilökohtaisia tietojani käsitellään luottamuksellisesti ja tutkimustulosten julkaisuissa tutkittavien tunnistaminen ei ole mahdollista.

Olen ymmärtänyt, että tutkimuksessa otettavista verinäytteistä tehdään tutkimuksen seurantajakson päätyttyä geenianalyysejä Kuopion yliopiston lääketieteellisessä tiedekunnassa sekä Jyväskylän yliopiston liikuntabiologian laitoksella. Näytteitä käytetään vain tutkimuksessa tarvittaviin selvityksiin koskien fyysisen kunnon, kuormituksen ja kehon koostumuksen välisiä yhteyksiä.

Vakuutan, että terveydentilani on hyvä ja että en ole minkään lääkehoidon alaisena. Lupaan viipymättä ilmoittaa tutkimushenkilökunnalle mikäli terveydentilassani on tapahtunut muutos tutkimusjakson aikana. Ymmärrän, että annettujen ohjeiden ja rajoitusten, kuten tutkimusta edeltävän paaston, tarkoituksena on varmistaa turvallisuutta, ja lupaan noudattaa kaikkia tutkijoiden ja tutkimuslääkäreiden antamia ohjeita. Hyväksyn sen, että tutkijalääkäri voi keskeyttää osallistumiseni suostumuksestani riippumatta.

_____ ___/___/2009 _____

tutkimushenkilön allekirjoitus

Tutkijan osuus:

Vakuutan, että olen antanut tutkittavalle ennen tämän asiakirjan allekirjoittamista riittävän selvityksen tutkittavan oikeuksista sekä tutkimukseen liittyvistä yksityiskohdista siten kuin lääketieteellisestä tutkimuksesta annetun lain 488/1999 6§:ssä edellytetään. Vakuutan, että kaikkea tutkimuksen aikana saatavaa tietoa käsitellään luottamuksellisesti ja että tutkimusryhmän ulkopuolisille annettavasta tiedosta (esim. julkaisut) tutkittavien henkilöllisyys ei ole tunnistettavissa. Tutkittavalla on oikeus milloin tahansa tutkimuksen kestäessä (myös syytä ilmoittamatta) peruuttaa suostumuksensa tutkimukseen, ilman että peruutus vaikuttaisi tutkittavan oikeuteen saada tarvitsemaansa hoitoa.

_____ ___/___/2009 _____

tutkijan allekirjoitus ja nimenselvennys

APPENDIX 2 Loading day measurement time table

7:15	Subject Arrives
7:20	BIA + Heart Rate (5min)
	Electrodes (R leg)
9:00	170 kcal: energy bar + water
10:00	PRE
	MVC + EMG + Blood lactate
10:50	Prep for loading: VO_2 mask, gear + Heart Rate (5min)
11:00	START COMBINED LOADING
12:00	POST IMMEDIATE
	MVC + EMG + Blood lactate + Heart Rate (1min)
12:05	PASSIVE OR ACTIVE RECOVERY
13:05	POST RECOVERY
	MVC + EMG + Blood lactate + Heart Rate (5min)
14:00	170 kcal: energy bar + water
15:00	Finished Loading Day

Next morning: Force + EMG + Blood lactate + Heart Rate (5min)

APPENDIX 3 Combined loading protocol

COMBINED LOADING PROTOCOL WORKSHEET

Subject ID#: _____ Subject Initials: _____ Birthdate: _____ Date: _____

Male Age: _____ yrs Ht: _____ cm Wt: _____ kg Total Wt: _____ kg

VO_2max: _____ $ml \cdot kg^{-1} \cdot min^{-1}$ HRmax: _____ bpm 1-RM: _____ kg 70% 1-RM: _____ kg

Seat back #: _____ Test start time: _____

1st set E : 10-min Treadmill						
Stage	Speed ($km \cdot h^{-1}$)	Time (min)	HR (bpm)	RPE	VO_2 ($ml \cdot kg \cdot min^{-1}$)	Comments
1	4.5	0-1				
		1-2				
		2-3				
		3-4				
		4-5				
2	7.0	5-6				
		6-7				
		7-8				
		8-9				
		9-10				
1st set S : 2 x 10 reps @ 70% 1-RM - 2 min rest after each set						
3	Set	10-14 min	Weight (kg)	Assisted Reps	Max Assisted Force (kg)	Comments
	1					
	2					
2nd set E : 10-min Treadmill						
4	4.5	0-1				
		1-2				
		2-3				
		3-4				
		4-5				
		5-6				
		6-7				
		7-8				
		8-9				
		9-10				

COMBINED LOADING PROTOCOL WORKSHEET

2nd set S : 1 x 10 reps @ 70% 1-RM - 2 min rest after each set						
5	Set	8-12 min	Weight (kg)	Assisted Reps	Max Assisted Force (kg)	Comments
	3					
3rd set E : 10-min Treadmill						
6	7.0	0-1				
		1-2				
		2-3				
		3-4				
		4-5				
7	4.5	5-6				
		6-7				
		7-8				
		8-9				
		9-10				
3rd set S : 2 x 10 reps @ 70% 1-RM - 2 min rest after each set						
8	Set	10-14 min	Weight (kg)	Assisted Reps	Max Assisted Force (kg)	Comments
	4					
	5					
End of C Protocol						

C End Time: _____ 1-min HRV Time Clock: _____, Watch: _____

APPENDIX 4 Active recovery protocol

C - ACTIVE RECOVERY WORKSHEET

Subject ID#: _____ Date: _____

C-AR start time: _____ 1-RM: _____ kg 30% 1-RM: _____ kg

S-AR Protocol: 5 x 10 reps @ 30% 1-RM - 5 min rest after each set									
E-AR Protocol: 30-min Treadmill (5:5)									
Stage	Speed ($km \cdot h^{-1}$)	Time (min)	HR (bpm)	RPE	Stage	Speed ($km \cdot h^{-1}$)	Time (min)	HR (bpm)	RPE
1	3.0	0-1			4	0	15-16		
		1-2					16-17		
		2-3					17-18		
		3-4					18-19		
		4-5					19-20		
2	0	5-6			5	3.0	20-21		
		6-7					21-22		
		7-8					22-23		
		8-9					23-24		
		9-10					24-25		
3	3.0	10-11			6	0	25-26		
		11-12					26-27		
		12-13					27-28		
		13-14					28-29		
		14-15					29-30		
End of C-AR Protocol									

C-AR end time: _____