ACUTE HIT SESSION INDUCED CHANGES AND RECOVERY IN MUSCLE ACTIVATION LEVEL, VOLUNTARY FORCE PRODUCTION AND JUMP PERFORMANCE DURING 8 WEEKS OF HIT TRAINING IN RECREATIONALLY ENDURANCE TRAINED MEN

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ABSTRACT

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High-intensity interval training (HIT) has gained wide popularity among competitive athletes and sport enthusiasts in the recent decades. While an extensive amount of research has been done on acute neuromuscular (NM) response and recovery after low-moderate intensity endurance training, the data regarding HIT in recreational athletes remains to be limited. In order to reduce that gap, the present study investigated the acute HIT session induced changes and recovery in muscle activation level, voluntary force production and counter movement jump performance during 8 weeks of HIT training in recreationally endurance trained men. A total of 9 subjects age 30.7 years (± 5.8), participated in the present study. The subject completed 8 weeks HIT training period (3-5 HIT sessions per week). NM measurements took place at weeks -1, 4 and post 8, respectively. The measurements included isometric unilateral knee extension with electrical stimulation and a counter movement jump (CMJ). NM measurements were performed before, after and 24 h after the HIT session at weeks -1, 4 and post 8, respectively. There was an acute decrease in maximal voluntary contraction (MVC) and muscle activation level (AL) following HIT sessions, while a significant decrease was observed only at week 4, being 12.3 % ± 8.6 for MVC and 8.2 % (± 7.9) for AL respectively. CMJ performance was significantly enhanced after the HIT session at weeks -1 (8.0 % ± 6.3) and 4 (7.7 % ± 6.8). MVC capacity was recovered within 24 hours post HIT and no potentiation of CMJ was any more present. The major findings of the study showed that isometric MVC and muscle AL of lower extremities can be significantly impaired after HIT, while jumping ability can be enhanced due to post activation potentiation (PAP). The magnitude of the changes is highly individual. However, in recovery of 24 hours post session PAP was not observed any more as well as MVC and AL impairment.

Key Words: High-intensity interval training, acute neuromuscular fatigue, CMJ, MVC, muscle AL, running performance, endurance athletes

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The present master thesis is a part of larger research project, led by Professor Keijo Häkkinen. The study was supported and funded by the department of Biology of Physical Activity. There will be a total of three master theses from this project. The present master thesis was focused on acute neuromuscular responses to high intensity interval training in endurance trained man. The author and research team would like to thank the Department of Biology of Physical Activity, its staff and students who actively helped along the research and especially my supervisor and dedicated subjects.

CONTENTS

1. INTRODUCTION

During muscle exercise, fatigue is defined as the inability to maintain the required level of strength (Edwards, 1981). As exercise intensity or duration (or both) increase, fatigue develops progressively until the muscle is no longer able to perform the requested task. Fatigue is a complex, multifactorial phenomenon whose mechanisms are influenced by the characteristics of the task being performed (i.e. type and duration of the exercise, speed and duration of the muscle contraction (Enoka and Stuart, 1992). Therefore, the magnitude and developmental patterns of fatigue would differ for exercises of different intensities.

In the context of endurance sports, training intensity can be categorized into 3 main broad categories: low intensity training, moderate intensity training and high intensity training. In the recent decades, high intensity training was under extensive research and a wide variety of investigations have been made in order to explain its mechanisms and benefits for performance, also a great number of application strategies were presented. Today, high-intensity interval training (HIT), in a variety of forms, is one of the most effective means of improving cardiorespiratory and metabolic function and, in turn, the physical performance of athletes. HIT involves repeated short-to-long bouts of rather high-intensity exercise interspersed with recovery periods (Buchheit and Laursen, 2013).

Nowadays, high intensity interval training (HIT) is widely used among competitive athletes and sport enthusiasts. While an extensive amount of research has been done on acute neuromuscular (NM) response and recovery after low-moderate intensity endurance training, the data regarding HIT in recreational athletes remains to be limited. Therefore, the purpose of the present study was to investigate the acute HIT session induced changes and recovery in muscle activation level, voluntary force production and counter movement jump performance during 8 weeks of HIT training in recreationally endurance trained men.

2. NEUROMUSCULAR FATIGUE

In common, ‘‘fatigue’’ is a term used to describe the decrease in physical performance associated with an increase in the real and/or perceived difficulty of a task or exercise (MacInstosh et al. 2005). During muscle exercise, fatigue is defined as the inability to maintain the required level of strength (Edwards, 1981). This definition is associated with the sudden appearance of fatigue and inability to sustain the exercise. However, fatigue is a complex phenomenon and its mechanisms are influenced by such parameters of the task as: type and duration of the exercise, and type, speed and duration of the muscle contraction (Enoka & Stuart, 1992). Many neurophysiological mechanisms are disturbed already before the body feels the effect of fatigue; these disturbances and their consequences serve as precursors of fatigue. Furthermore, the initial state of the neuromuscular system - energy reserves, ion concentrations and the arrangement of contractile proteins is altered as soon as exercise starts. Fatigue then develops progressively until the muscle is no longer able to perform the requested task. Therefore, neuromuscular fatigue can be defined as ‘‘any exercise induced reduction in force or power regardless of whether the task can be sustained or not” (Bigland-Ritchie and Woods, 1984).

Many different factors may be involved in the expression of neuromuscular fatigue. The maintenance of submaximal strength over time results from facilitatory and inhibitory influences of neuromuscular origin. In fact, the neuromuscular system tries to compensate for the decrease in force generation by implementing a variety of nervous and muscle-related mechanisms, in order to delay the point at which the task can no longer be performed.

During maximal or submaximal muscle exercise, fatigue is caused by several different physiological reasons. Scientific literature distinguishes two main types of fatigue – central and peripheral. “Central fatigue” comprises a decrease in voluntary activation of the muscle, meaning a decrease in the number and discharge rates of the motor units (MUs) recruited at the start of muscle force generation, while ‘‘peripheral fatigue’’ indicates a decrease in the contractile strength of the muscle fibres and changes in the mechanisms underlying the transmission of muscle action potentials (Gandevia, 2001).

These phenomena occur at the nerve endings and at the neuromuscular junction (NMJ) and are usually associated with peripheral fatigue. The set of sites which can lead to a decrease in force generation is presented on the Figure 1.

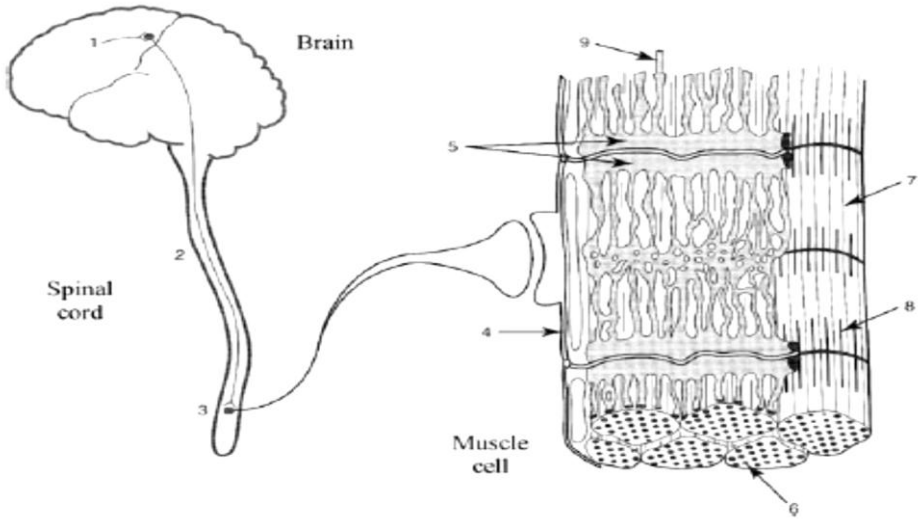

FIGURE 1. The sites of neuromuscular fatigue (Obtained from Boyas and Guevel, 2011).

According to the figure, the following sites can contribute to neuromuscular fatigue: (1) activation of the primary motor cortex; (2) propagation of the command from the central nervous system (CNS) to the motoneurons; (3) activation of the MUs and muscles; (4) neuromuscular propagation (including propagation at the NMJ); (5) excitation-contraction coupling; (6) availability of metabolic substrates; (7) state of the intracellular medium; (8) performance of the contractile apparatus; (9) blood flow. Sites 1-3 are associated with central fatigue, while sites 4-9 are associated with appearance of peripheral fatigue.

Several attempts were made in order to understand the mechanisms and physiological sites that can be affected by neuromuscular fatigue and thus may be the source of a decrease in force generation. According to Boyas and Guevel (2011), central fatigue mainly involves blocking of action potentials propagation at axonal branching sites, inducing a loss of activation of the muscle fibers. In addition, muscle afferents (neuromuscular spindles and Golgi tendon organs) may impair the activity of motoneuron, as well as the stimulation of type III and IV nerves may induce a drop in the motoneuron discharge rate and an inhibition of motor cortex command. Lastly, the

exercise could lead to the release of cytokines such as interleukin-6, which is associated with the sensation of fatigue. The physiological mechanisms of peripheral fatigue differ from those of central fatigue; they are presented in the table 1.

TABLE 1. Sites and physiological mechanisms potentially affected by peripheral fatigue (modified from Boyas and Guevel, 2011).

Changes within the muscle fibers	**Changes in the intracellular environment**
Accumulation of inorganic phosphate and hydrogen ions in the sarcoplasma, inducing a drop in the contractile force due to inhibition of the cross-bridges' interactions	Accumulation of lactate and hydrogen ions**.** The accumulation of hydrogen ions is partly buffered by bicarbonate, which induces a release of carbon dioxide. This changes the respiratory quotient
Inhibition of the calcium release by the SR	Accumulation of ammonia
Accumulation of magnesium ions in the sarcoplasma. The magnesium ions may limit calcium release by the SR	Accumulation of heat, which induces greater sweat secretion. The water loss associated with this phenomenon could lead to dehydration
A decrease in glycogen reserves and (in extreme cases) a drop in blood glucose.	
Drop in the nerve action potentials' speed of propagation along the sarcolemma	
Increase in the efflux of potassium ions from the muscle fibres. The increase in potassium in the lumen of the transverse tubules could block action potentials at this point and hence diminish the force generation	

2.1 Central Fatigue

Central fatigue can be defined as a reduction in the maximal capacity of the central nervous system to optimally recruit motor units to produce force (Gandevia, 2001). Central fatigue comprises all supraspinal and spinal physiological phenomena capable of inducing a decrease in motoneuron excitation. The presence of central fatigue can be evaluated by using percutaneous electrical stimulation (Shield and Zhou, 2004). During maximal voluntary contraction (MVC), the observation of a force peak following an electrical stimulation superimposed on the motor nerve innervating the muscle indicates that the voluntary activation was not in fact maximal. Meaning that some MUs are either not recruited or do not fire often enough to allow the muscle fibers to generate maximal force. This in turn would lead to progressive impairment of voluntary activation and to the development of central fatigue.

Several transcranial magnetic stimulation (TMS) studies by Gandevia and colleagues have shown that central fatigue can account for over 25% of the drop in force seen during sustained, maximal contractions (Gandevia, 2001; McNeil and Gandevia, 2009; Taylor and Gandevia, 2008). Interestingly, it was observed that central fatigue appears to contribute more significantly to the decrease in force generation during low-intensity, long duration exercises (Millet and Lepers, 2004). The existing data shows that one of the causes of weaker central command during prolonged exercise could be the decreased excitation supplied by the motor cortex also known as “supraspinal fatigue” (Taylor et al. 2006).

The causes of supraspinal fatigue are poorly known. However, several explanatory hypotheses have been suggested. Firstly, supraspinal fatigue may be linked to the depletion or accumulation of certain brain neurotransmitters, inducing a decrease in cortico-spinal descending excitation. The most studied neurotransmitter in this context is serotonin (5-hydroxytryptamine [5-HT]). Newsholme et al. (1987) were among the first to show that prolonged exercise increased the brain’s serotoninergic activity, which in turn limits central command and thus, the recruitment of motor units. Central fatigue could also be influenced by the activity of other neurotransmitters. More generally, various catecholamines (e.g. adrenaline, noradrenaline and dopamine) may have an

effect on fatigue by virtue of their effect on motivation and motor action (Meeusen and Meirleir, 1995).

Furthermore, central command may be limited by changes in the brain concentration of other substances, such as ammonium ions and glycogen (Boyas and Guevel, 2011). Several investigations have shown that during exercise, the plasma concentration of ammonium increases and ammonium ions can easily cross the blood-brain barrier, so their accumulation could be a factor in the drop in motor cortex activity during prolonged exercise, via its effects on cerebral brain blood flow, the activity of certain neurotransmitters and synaptic transmission (Nybo et al. 2005). Glycogen could also play a role in central fatigue because brain activation is associated with a drop in brain glycogen. The influence of the brain's neurotransmitter, ammonium and glycogen levels on central fatigue and performance has been evidenced in prolonged exercise over 30 minutes (Nybo et al. 2003).

Another hypothesis concerning supraspinal fatigue suggests that certain muscle afferents related to the muscles' biochemical status and force generation capacity limit cortical activity. In a comprehensive survey of the literature, S.C. Gandevia (2001) stated that: "Feedback on the muscles' biochemical status and force generation capacity is likely to reduce stimulation from cortical sites. It is, therefore, possible that the fatigue sensitive muscle afferents limit voluntary command by acting upstream of the motor cortex" (Gandevia, 2001).

The assumption made by S.C.Gandevia was supported by recently proposed hypothesis. It states that the exercise performance is regulated by sensory feedback to the central nervous system to ensure that an individual's critical threshold of peripheral fatigue is never exceeded. This hypothesis predicts that high levels of peripheral fatigue regulate exercise performance by limiting the extent of skeletal muscle recruitment through afferent feedback from the exercising muscles to the central nervous system (Amann, 2011, 2012) (see figure 2). Indeed, the inhibitory afferents from intramuscular receptors appear to be involved in the decrease in motoneuron activity. Stimulation of group III and IV muscle afferents (metaboreceptors) during fatiguing exercise was shown to inhibit the activity of the alpha motoneurons (Martin et al. 2006). The neuromuscular spindles (group Ia and II afferents) may be also involved in fatigue development. It was

shown that the discharge rate of these receptors decreases progressively during isometric muscle contractions. The drop in discharge rate induced by neuromuscular spindles may thus be associated with the limitation of alpha motoneuron activity. Nevertheless the influence of neuromuscular spindles on muscle's contractile activity remains to be determined.

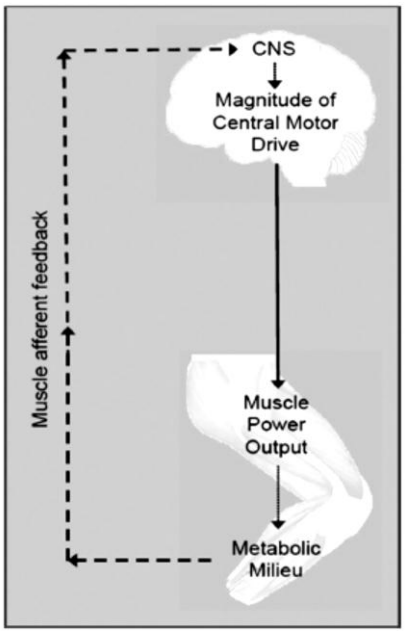

FIGURE 2. The scheme of performance regulation by sensory feedback to the central nervous system (Amann, 2011)

The Golgi tendon organs (group Ib afferents) at the musculotendinous and musculoaponeurotic junctions provide the CNS with feedback on the intramuscular tension. These mechanoreceptors are thought to inhibit neuronal activity. The Golgi tendon organs' exact effects on motoneuron activity during a fatiguing task are difficult to determine because these afferents are difficult to isolate and their projections include interneurons receiving signals from Ia afferents (Boyas and Guevel, 2011). The influence of the signals emitted by the neuromuscular spindles and the Golgi tendon organs has hardly been studied and is poorly characterized – mainly because their effects can be attenuated at the spinal level and is less intense than those of many group III and IV afferents (Boyas and Guevel, 2011). However, it is clear that many anatomic sites and physiological mechanisms are affected by central neuromuscular fatigue. This type of fatigue also appears to perturb normal processes in the peripheral part of the neuromuscular system.

2.2 Peripheral Fatigue

Peripheral fatigue is defined as the reduction in force originating from the sites at or distal to the neuromuscular junction (Gandevia, 2001). The factors involved in peripheral fatigue include alterations in neuromuscular transmission, muscle action potential propagation, excitation-contraction coupling and related contractile mechanisms. Neuromuscular transmission is defined as transformation of the nerve action potential (AP) into a muscle action potential and takes place at the neuromuscular junction (Boyas and Guevel, 2011). During fatigue, this mechanism can be altered by: (1) insufficient propagation of the nerve potential at the nerve endings; (2) a failure of the coupling between excitation and neurotransmitter secretion in the synaptic gap; (3) neurotransmitter depletion; (4) reduced neurotransmitter release; (5) a decrease in the sensitivity of the post-synaptic acetylcholine receptors and the post-synaptic membrane (Allen et al. 2008).

During sustained contraction, fatigue may decrease the excitability of small-diameter axons. A slight decrease in excitation may then lead to inactivation of these axons and a decrease in the amount of neurotransmitter released in the synaptic gap leading to decreased force production capacity.

Of the many metabolic changes associated with prolonged muscle contraction, two in particular appear to underlie the myofibril's reduced force generation capacity during fatigue - the increase in intracellular concentrations of hydrogen ions (H^+) and inorganic phosphate (Pi). The Pi concentration increases during exercise, due to: dissociation of phosphocreatine into Pi and creatine and hydrolysis of adenosine triphosphate (ATP). Hydrogen ions accumulate as a result of the hydrolysis of ATP and the production of lactic acid which occurs when glycolytic activity exceeds the mitochondria's oxidative capacity or when the oxygen supply is limited.

The accumulation of H^+ has long been considered to be a significant cause of the decrease in myofibrillar force generation, since it is associated with a decrease in intracellular pH, which perturbs chemical reactions. Nevertheless, these results were obtained at sub-physiological temperatures. In fact, Westerblad and co-workers already

in 1997 have shown that the acidification of the medium had a very limited effect on the force generation capacities at normal body temperatures (Westerblad et al. 1997). Therefore, if the acidosis is not the cause of the drop in force generation, then the accumulation of Pi must be.

Inorganic phosphate could perturb force generation by decreasing the myofibrils' sensitivity to Ca2+ and by acting directly on the cross-bridges. The increase in the Pi concentration under fatiguing conditions may perturb the cross-bridges' contraction-relaxation cycles. Taken as a whole, these facts suggest that the decrease in force generation observed at the very start of contraction (when the intracellular Ca2+ concentration is high) may be due to the accumulation of Pi (Boyas and Guevel, 2011). When muscle work is sustained, the drop in force generation may be also related to a decrease in the quantity of Ca2+ released by the sarcoplasmic reticulum (SR). This phenomenon may occur later in fibrils with strong oxidative capacities and sooner in fatigued fibres under anaerobic conditions. Variations in Pi and ATP concentrations appear to be responsible for the impaired release of Ca2+ (Boyas and Guevel, 2011).

Summarizing the above mentioned, the present data suggests that the drop in force generation during sustained exercise is mainly related to a drop in the quantity of Ca2+ released by the SR. This may be primarily due to the intracellular accumulation of Pi and the decrease in ATP reserves associated with fatiguing exercise. However, it is important to remember that during sustained exercise, the blood supply and reserves of metabolic substrates are also likely to influence performance.

During muscle contraction, a reduction in blood flow was one of the first mechanisms to have been identified as being involved in fatigue (Boyas and Guevel, 2011). During physical activity, an increase in blood flow is necessary to supply the active muscles with substrates, evacuate metabolites and dissipate heat. However, muscle contraction often compresses the blood vessels and thus decreases the blood supply to the active muscles. Limitation of the blood supply will reduce the oxygen supply and promote the activity of anaerobic metabolic pathways. Additionally, the drop in blood flow will trigger more rapid accumulation of the metabolites associated with muscle contraction (e.g. Pi, H+) and will thus accelerate the process of fatigue and the drop in force

generation. Furthermore, the greater the contraction intensity, the earlier ischaemia will occur (Crenshaw et al. 2000).

In most muscle contractions, metabolic substrate availability does not constitute a performance-limiting factor. However, during certain types of endurance exercise (such as cycling at submaximal intensity, i.e. 70 to 80% of the maximal aerobic power), the inability to maintain the required force appears to coincide with the depletion of the leg extensors' glycogen reserves. Intake of additional glucose enables the subjects to maintain the exercise for longer (Boyas and Guevel, 2011).

Taken together, central fatigue involves impairment of voluntary muscle activation and seems to occur particularly during submaximal, low-intensity muscle contractions. It can be due to a decrease in the excitation supplied by the motor cortex and/or a decrease in motoneuron activity. The activities of the brain's neurotransmitters and muscle afferents appear to be prime factors in the occurrence of central fatigue, which occurs particularly when the triggering exercise is low intensity. Peripheral fatigue corresponds to an alteration in muscle contraction. It can be induced by perturbations of: (1) neuromuscular transmission; (2) the propagation of the muscle action potential; (3) excitation-contraction coupling and (4) perturbations of contractile mechanisms.

During prolonged muscle contraction, the body has several mechanisms for maintaining the level of force produced and resisting fatigue. New motor units will be recruited to compensate for the fatigue of those activated at the start of the contraction. Fatigue can also be considered as a mechanism for limiting the harmful effects of exhausting muscle exercise. The muscle afferents provide the nervous system with information on the state of the muscles and appear to be involved in the regulation of fatiguing exercise.

Lastly, the appearance of neuromuscular fatigue is associated with changes in several of the above mentioned mechanisms, whether central or peripheral. In whole-body exercises, central fatigue is believed to occur after peripheral fatigue has already developed and to be dependent on exercise duration. Interactions between central and peripheral fatigue lead to a cascade of events which accelerate or decrease the muscle's force generation capacity.

3. NEUROMUSCULAR RESPONSE AND ADAPTATIONS TO ENDURANCE TRAINING

Regardless of the locomotion type and intensity the neuromuscular system is essential for human locomotion and is made up of central and peripheral mechanisms including the brain and nerves (central) and muscles (peripheral). During physical activity the neuromuscular system gets stressed which in turn leads to the appearance of neuromuscular fatigue. Neuromuscular fatigue can be measured as a reduction in maximal voluntary contraction (MVC) force. Generally endurance exercises can be performed in a form of low intensity (LIT) and high intensity (HIT) training. It is well established that both neuromuscular and peripheral fatigue increase with increasing intensity during exercise bouts of a similar duration (Froyd et al. 2016).

The causes of fatigue after different types of exercise (i.e., task dependency) are now better understood. Substantial reductions of MVC levels and evoked peak force response (as an index of peripheral fatigue) occur after approximately 20% of the total duration of a self-paced time trail (TT) exercise with further increases as the exercise continues (Froyd et al.2013). Some attempts have been made to determine the extent to which different aspects of fatigue develop during self-paced exercise of different durations. A recent study by Thomas et al. (2015) observed greater levels of central fatigue after 40-km cycling TT and 20-km cycling TT compared with 4-km cycling TT, but a higher level of peripheral fatigue after the 4-km TT compared with the 20-km TT and 40-km TT, indicating that the mechanisms of neuromuscular response as well as the time course and magnitude of fatigue, depend on the duration and intensity of the exercise.

The existing data shows that the extent to which peripheral and central processes contribute to fatigue is dependent on the nature of the task, and hence, task dependency remains a central theme in the study of fatigue. Different training modes can be considered as endurance or aerobic exercises, comprising such training modes as walking, Nordic-waling, uphill/stairs climbing, cycling, running, skiing, rowing and other cyclic locomotion types. Additionally, several regimes of resistance training can stress the cardiovascular system and improve aerobic capacity in a similar manner as

aerobic exercises. Those regimes are: body weight training, circuit-training and other low load-high volume types of strength training. An extensive research on endurance training has clearly shown that aerobic exercises performed with sufficient duration and/or intensity are able to stress the cardiorespiratory, cardiovascular and neuromuscular systems even though the locomotion patterns of those exercises can be different. The differences in movement patterns would in turn lead to difference in neuromuscular responses to each mode of exercises.

Nowadays it is accepted that endurance exercises lead to a smaller magnitude of neuromuscular responses compare to the response observed following strength exercise. For example, the study of Paavolainen and colleagues (1999) observed large decreases in strength and neural activation following strength exercise, while only small-scale decreases in strength and neural activation were observed following running sessions (Paavolainen et al. 1999). However, it is important to note that the magnitude of fatigue is dependent on the intensity and /or duration of the exercise. Indeed, marathon and ultra-marathon running or skiing was shown to cause a significant amount of fatigue (Nicol et al 1991; Millet et al. 2003; Millet et al. 2004; Millet, 2011).

Lower neuromuscular fatigue observed in running can be partly explained by the fact that endurance running typically requires less force production than strength exercise. It was observed that even maximal uphill running cannot induce maximal muscle activation (Sloniger et al. 1997). On the other hand, running involves repetitive force production and usage of stretch shortening cycle (SSC) which can be quite demanding both physically and metabolically, thus leading to above mentioned central and peripheral fatigue. For example, it was shown that after 30 km of high intensity trail running the decrease in maximal voluntary contraction (MVC) was 24% (Millet al. 2003). After simulated marathon racing a 22% decrease in MVC and 16% decrease in drop jump were observed (Nicol et al. 1991), while after a 5 km time-trial the MVC of the lower extremities has been shown to decrease by 15% (Nummela et al. 2008).

There could be few explanations behind the observed decreasement in MVC. It was suggested that the decreases in isometric and dynamic performance are partly the result of changes in muscle stiffness, reduced economy/efficiency and a reduced ability to use elastic energy, indicating that repetitive stretch-shortening of muscles during running

actions is able to cause significant fatigue. Indeed, Avela and Komi already in 1998 observed that alternations in muscle stiffness may affect the development of fatigue. Although it appears that strength loss is highly correlated with decreases in voluntary muscle activation (Millet et al. 2003), however, the average decreases in MVC are not necessarily related to decreases in running velocity during a running trial (Paavolainen et al. 1999; Nummela et al. 2008).

Another issue that can significantly influence the magnitude of neuromuscular fatigue following an endurance training session is individual's training status. For example, Vuorima et al. (2006) has shown that trained middle and long distance runners who completed intensive running for 20-40 min have increased their jump and half-squat performance following the session, suggesting that under certain circumstances an endurance training session may cause potentiation.

The individual's training status also has a significant effect on recovery. It appears that at least 48 h are needed after a 10 km running at race pace in trained runners to fully recover neuromuscular characteristics (Gomez et al. 2002). A greater amount of time may be necessary to recover from longer distances or intensities of running due to accumulation of greater amount of muscle damage from repetitive stretch shortening cycle actions (Taipale et al. 2013).

Taken as a whole, the existing data suggests that the magnitude of neuromuscular fatigue following an endurance exercise is dependent on training duration and intensity as well as from subject's training status. Fatigue after endurance exercises might be more peripheral than central in nature. However, it is difficult to separate them, since the mechanisms of both central and peripheral fatigue seem to be activated by endurance exercises.

3.1 Neuromuscular adaptations to endurance training

Greater the amount of neuromuscular response, greater the observed neuromuscular adaptations would be. In line with that, neuromuscular adaptations to an endurance exercise session are typically not so pronounced compare to those observed following

strength exercise due to lower magnitude of neuromuscular response to endurance training compare to a strength training session.

The mode of endurance training has a significant influence on adaptations since they are specific to the movement patterns performed. It was shown that different modes of endurance exercises induce "task-dependent" fatigue and affect muscle damage in different ways (Millet and Lepers, 2004; Millet et al. 2009). Neuromuscular adaptations appear to be specific to the force and velocity of the movements performed meaning that athletes involved in different types of training will have different neuromuscular adaptations. For example, studies that compared sprinters to distance runners found different adaptation patterns in those groups. It was shown that trained strength athletes and sprinters are able to produce a greater absolute maximal force and higher maximal force per unit of cross-sectional area than endurance athletes, indicating training specific neuromuscular adaptations (Häkkinen and Keskinen 1989).

Typical neuromuscular adaptations observed after an endurance training period in previously untrained subjects include changes in the size of the muscle, contractile properties of fibers, changes in fiber type prevalence towards oxidative fibers (due to mitochondrial growth) and an enhanced recruitment of motor units in active muscles (Luden et al.2012). Human muscles are highly plastic; however, there are not many studies that specifically examine the neuromuscular adaptations to running, because controlling for running technique (foot strike patterns), footwear, and daily physical activity is quite difficult. Another mode of endurance training widely used in research purposes is cycling. For example, it was shown that 10 weeks of cycling induces increases in strength of the lower extremities in untrained males (Farup et al. 2012). Lucia et al. (2000) has also shown that endurance cycling enhances the recruitment of motor units in the active muscles.

3.2. Neuromuscular responses and adaptations to high intensity interval training (HIT)

High-intensity interval training (HIT) is defined as either repeated short (<45 s) to long (2 - 4 min) bouts of rather high- but not maximal-intensity exercise, or short (<10 s, repeated-sprint sequences [RSS]) or long (>20–30 s, sprint interval session [SIT]) all-

out sprints, interspersed with recovery periods (Buchheit and Laursen, 2013). Any HIT training session will challenge, at different respective levels relative to the training content, both the metabolic, the neuromuscular and musculoskeletal systems

The experiments have shown that different HIT designs can lead to the same cardiorespiratory response and adaptations but this does not apply for the neuromuscular system responses since different neuromuscular loads and anaerobic pathway contributions are involved, depending on the HIT protocol (see Table 2).

TABLE 2. The Effects of different run-based high-intensity interval training protocols on acute neuromuscular performance (modified from Buchheit and Laursen, 2013).

Format	**Work duration**	**Work intensity**	**Relief duration**	**Relief intensity**	**Acute change in muscular performancec**
Game based training	> 2 – 3 min	< 95 % vVO2max	< 2 min	55 % vVO2max	From improved to impaired
HIT with Long intervals	> 2 – 3 min	95 % or > 95 % vVO2max	2 – 4 min	60–70 % v V O2max / Passive	From improved to impaired
HIT with Short intervals	< 20 sec	100 – 120 % vVO2max	< 20 sec	Passive	From improved to impaired
Repeated sprints	< 5 sec	All out	< 30 sec	55 % vVO2max to passive	Impaired to slightly improved
Sprint interval training	> 20 sec	All out	> 2 min	Passive	Impaired to significantly impaired

vVO2max - lower speed associated with maximal oxygen uptake

The acute neuromuscular load/musculoskeletal strain associated with HIT sessions should also be considered with respect to long-term performance development, the

possible interference with other training content, as well as acute and chronic injury risk. An appropriate planning is of importance since it was shown that neuromuscular fatigue, if maintained for several hours/days after the HIT session, can have a direct effect on the 'quality' of subsequent training sessions (Bompa and Haff, 2009). The residual neuromuscular fatigue post-HIT may reduce force production capacity and rate of force application during the following (strength /speed) sessions, which can, in turn, attenuate training stimuli for optimal neuromuscular adaptations. However, it should be noted that the acute effects of high-intensity running on performance depend on the physiological characteristics and training history of the athlete.

The existing data on neuromuscular response to HIT suggests that fatigue induced by HIT including either very short (<20 s) to short (<1 min) and/or non-maximal efforts (<120 % of v V O2max) tends to be predominantly peripheral in origin i.e. alterations to muscle excitability and excitation-contraction coupling, related to intramuscular potassium concentration disturbance and accumulation of metabolic by-products including inorganic phosphate and hydrogen ions (Perrey et al. 2010). On the other hand, it was shown that performance impairment during repeated long (>30 s) and all-out sprints may be more essentially related to central fatigue (Fernandez et al.2013).

While direct comparisons between long- and short-bout HIT have still to be documented, the acute neuromuscular load may be greater with short intervals for the following reasons; first, work intensity is generally higher with shorter intervals. While the majority of muscle fibers might already be recruited during long intervals the firing rate and relative force development per fiber is likely greater during short intervals. Second, short intervals require frequent accelerations, decelerations and re-accelerations. In addition to the increased metabolic and muscle force demands during acceleration phases of high-intensity exercise the completion of short intervals requires achievement of a greater absolute speed (Buchheit and Laursen, 2013).

In case of short duration (10- 20 sec) repeated-sprint sequences (RSS) training, especially when RSS are repeated within a short time period of 2–5 min, neuromuscular fatigue, as evaluated from post-exercise jump tests, is accentuated. To implement 'quality' RSS sessions a prolonged and likely active recovery period up to 15-20 min should be implemented between sets to maximize muscle recovery (Buchheit, 2012).

For Long duration (>20-30sec) sprint interval sessions (SIT) the large speed or power decrement score from 5 to 20 % is generally observed, suggesting large neuromuscular function impairment following a SIT session. Recent data suggests that in contrast to other forms of HIT, central mechanisms may be the primary origin of the impairment to MVC performance following the repetition of long sprints (Fernandez et al.2013).

It remains difficult to be certain on the acute effects that different HIT formats have on musculoskeletal and neuromuscular load of the athletes due to great variance in subjects training status and HIT protocols between studies. However, there is a clear trend showing that endurance-trained athletes generally perform long-bout HIT while team sports players usually perform short-interval HIT and RSS training. There is a bell-shaped relationship between the intensity of an HIT session and the acute neuromuscular performance, with too low and too high (all-out) intensities having not enough and acute detrimental effects, respectively (Fig. 3).

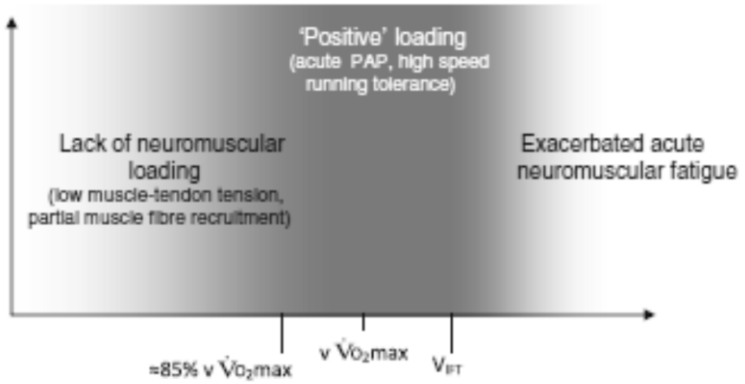

FIGURE 3. The relationship between the intensity of a HIT session and acute neuromuscular performance (Buchheit and Laursen, 2013)

Neuromuscular adaptations to HIT are also intensity specific. For example, it was shown that work intensities 80–85 % vVO2max require recruitment of fast twitch fibers, induce post-activation potentiation and possibly lead to long-term structural adaptations that allow fatigue-resistance to high-speed. In contrast, supramaximal-to-maximal (>120 % vVO2max) intensity exercises are likely associated with acute impairments in muscular performance and might even lead to maladaptations when training load is inappropriate (Buchheit and Laursen, 2013). According to the existing data, the optimum window for adaptation is somewhere between 85 – 100 % of vVO2max (Fig. 3).

3.3 Neuromuscular recovery after endurance and high intensity interval training

Long-distance racing of distances 20 km and greater have been reported to significantly decrease muscle strength and/or exercise capacity (i.e., multiple isokinetic repetitions, such as 50–60 repetitions) following competition (Gomez et al. 2002). This may be because of both muscle damage and glycogen depletion, which would be greater in the longer distances. With the marathon distance, Sherman et al. (1985) reported significant reductions in isokinetic strength and exercise capacity immediately after a marathon race. Interestingly, these measures did not return to baseline value until 3 days post-marathon. In the case of 20km simulated run, a decreases in area under the peak torque curve were observed, while maximal peak torque in the knee flexor / extensors were not affected (Kramer et al 1991). The authors speculated that the 20-km race did not significantly affect the fast-twitch motor units responsible for peak torque whereas the reduction in the area under the peak torque curve was due to the fatigue of the slow motor units.

Later it was hypothesized that with the faster speeds, higher threshold motor units would be recruited to run the race leading to pronounced impairments in strength and power capabilities already after 10 km distances. Gomes et al. (2002) investigated the effect of 10-km race on force production capabilities of muscle and the pattern of muscular recovery over 48 hours after the race. The main finding of the study was that only muscle force production in the hamstring muscle group was primarily affected, they have also showed that most of the force and power measures returned to baseline values within 48 hours after the race.

High intensity intermittent sessions of lower duration were also shown to cause fatigue and post session impairments in strength and power capabilities. Millet et al. (2004) investigated the acute neuromuscular response and recovery following highly strenuous uphill running exercise. They observed 8 % decreasement in isometric maximal voluntary contraction (MVIC) of knee extensor/flexor muscles compare to pre exercise performance (Figure 4). They have also shown that 45min after this type of exercise MVIC was lower than at the end of the exercise despite some recovery of excitation-

contraction coupling and no change in central activation. They concluded that MVIC capacity after strenuous HIT running is not fully recovered within 1 hour post exercise.

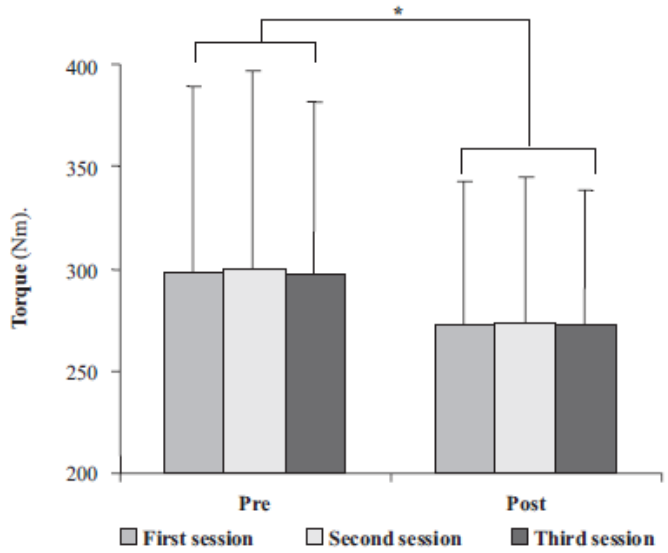

FIGURE 4. Mean values of maximal voluntary contraction pre and post high intensity exercise (Millet et al, 2004)

Few attempts were made to investigate the acute neuromuscular response and recovery patterns after high intensity short duration repeated sprint sequence training protocols (RSS). It was shown that a six-day HIIT program induced significant changes in repeated sprint ability and counter movement jump performance showing a temporary decline and a return to baseline level after 72 h of recovery (Wiewelhove et al. 2015). The authors suggested that since HIIT has the potential to induce muscle damage, it appears that the decreases in vertical jump height, jump efficiency and sprint performance may be related to repeated structural damage and inflammatory response of the muscle fibers caused by the HIIT program.

On the other hand, some studies have shown that neuromuscular performance after HIT can recover rather fast. Froyd et al. (2013) have shown that the major changes in neuromuscular function occur within the first 40% of exercise that is, for example, within the first 2 min of a ~6min exercise bout. They also observed that significant recovery occurs in skeletal muscle function within the first 2 min after exercise cessation, suggesting that the extent of peripheral fatigue has been underestimated by previous studies that investigated peripheral fatigue. However, none of any

neuromuscular function variables had returned to the pre-exercise value within 8min of rest.

The existing data on neuromuscular fatigue following endurance and HIT protocols is limited and many training protocols still require investigation. However, many studies observed great variety in the acute responses and recovery patterns following training, due to variations in exercise protocols and training status of the athletes. It is important to remember that responses to HIT and levels of fatigue as well as recovery patterns appear to be highly individual. As a general recommendation, the existing literature suggests to have at least 48h between strenuous endurance or HIT loadings to minimize the risk of chronic neuromuscular overload and injuries.

3.4 Neuromuscular response to strength training

Both endurance and strength training can lead to neuromuscular fatigue. Comparing to endurance exercise, already a single bout of strength training was shown to induce a large decrease in maximal force production capacity (McCaulley et al. 2009). However, it is important to understand that the magnitude and source of neuromuscular fatigue may vary depending on training volume, intensity, rest intervals, muscle action type (concentric, eccentric, and isometric), range of motion and time under tension (Raeder et al. 2016). Fatigue following a training session can result either from central factors or from peripheral factors. Central factors involve decrease in recruitment of new motor units and reduced firing frequency of the currently active units (Gandevia, 2001). Peripheral factors incorporate failure of force generation by the muscle, due to metabolic by-product accumulation and decreased excitation-contraction coupling (Bigland-Ritchie et al. 1986). However, fatigue can also results from a combination of those central and peripheral factors.

The magnitude and type of fatigue is highly dependent on the type of training performed. Strength training can be classified into several training regimes: hypertrophy (H), strength (S), and power (P) type resistance exercise (RE). Hypertrophy training incorporates large muscle groups at intensities of 70–80% of one repetition maximum (1RM), volumes of three sets of 10–12 repetitions, and rest periods of short duration (60–90 s) (McCaulley et al. 2009). Strength type RE includes higher intensities (85–

90% 1RM), lower volumes (3–5 sets of 3–5 repetitions) and extended rest periods (3–5 min) (Willardson et al. 2006). Compare to other types of RE, power type RE, incorporates high velocity total body movements such as jumps and squats with lower intensity (0–30% 1RM) and significant volumes (3–5 sets of 6 repetitions) followed by moderate rest periods (3 min) (Cormie et al. 2007). It is important to note that several regimes could be combined within one training session.

In general, loadings incorporating great neuromuscular activation will induce fatigue more quickly than less strenuous loadings (Häkkinen et al. 1988). Maximal strength training performed with high loads and low number of repetitions per set was shown to decrease muscle activation and to increase lactate accumulation leading to a significant acute fatigue (Ahtiainen et al. 2003). Indeed, strength type of RE is known to stimulate type II muscle fibers as well as increase motor unit activation and, therefore, may optimize the training stimulus to the nervous system and result in significant central fatigue (McCaulley et al. 2009). In contrast, power/explosive types of resistance training performed with low to medium loads (such as 30–60 % 1RM) and high velocity are not associated with high levels of blood lactate, however the studies have shown that such training mode can still induce acute fatigue and decreased muscle activation (Mikkola et al. 2007). It was also shown that the participation in a single high-intensity strength training session leads to a transient decline in neural activation, muscular force, and jump performance because of central and peripheral fatigue mechanisms (Raeder et al.2016). Lastly, RE performed with relatively high loads (such as 70-80%) for a moderate number of repetitions (e.g. 8-12) followed by short periods of rest (e.g. 1-2 minutes) i.e. hypertrophic training was shown to induce significant increase in blood lactate accumulation as well as acute neuromuscular fatigue (McCaulley et al. 2009). If several regimes are combined within one training session the magnitude and type of fatigue would be dependent on the chosen regimes, their order, training volume and intensity (see part 3.6 for more information).

Another type of strength training incorporates body weight training and/or circuit strength training performed with relatively low loads (such as 0-30 % 1RM) those types can be also called as muscle endurance training. This mode of training requires relatively low force production and causes less fatigue than maximal or explosive type resistance training protocols. However, there is evidence that this type of training can be

quite strenuous when performed with sufficient intensity and may lead to the similar responses as high intensity endurance training i.e. it may lead to significant NM fatigue (Klika and Jordan 2013).

The time needed for recovery is generally dependent on the intensity and duration of the training session. The time-course of recovery from an acute strength training session is important to know for optimizing training and reducing the chances for injury or overtraining. It was observed that after high intensity strength training it takes approximately 48 h to recover the force production capabilities in both trained and untrained individuals (Ahtiainen et al. 2004).

3.5 Neuromuscular adaptations to strength training

As well as endurance training strength training performed over a prolonged period of time leads to certain neuromuscular adaptations. In untrained individuals early-phase adaptations may be observed in already after 4-12 weeks of regular training, importantly untrained individuals are able to make more substantial gains in strength within a relatively short period of time compare to trained individuals, indicating that individual's training status has a significant impact on magnitude of time-course of adaptations (Häkkinen et al. 1985a; Häkkinen et al. 1985b).

It appears that over a more prolonged period of time the type of strength training performed may have a more marked and specific influence on neuromuscular adaptations than short-term training. Typical neuromuscular adaptations to RE are: (1) Synchronization and recruitment of additional motor units; (2) reduction of co-activation of agonist and antagonist muscles; (3) increases in motor unit firing frequency; (4) changes in the discharge rates of motor units and (5) increased neurotransmitter release at neuromuscular junction (Moritani and de Vries, 1979). Importantly, different types of RE may favor different adaptations. For example, maximal strength training typically results in early-phase neural adaptations that may be accompanied by muscle hypertrophy after prolonged training (Häkkinen, 1994). Similarly, explosive strength training results in specific neural adaptations that are evidenced by increases in muscle activation and especially rapid activation of the muscles due to increased motor unit recruitment. Explosive strength training may also

induce modest gains in maximal strength due to increases in motor unit firing frequency and increased motor unit recruitment (Häkkinen et al. 1985b).

Endurance athletes do not typically use hypertrophic strength training, however under certain circumstances it can be of great benefit for performance. Hypertrophic strength training causes significant increases in muscle cross-sectional area as well as neural adaptations (Moritani and de Vries, 1979). Circuit training with body weight as a load (muscle endurance) was also shown to induce changes in muscle activation but the changes are not as large in magnitude as those produced by maximal, explosive or hypertrophic training. These appear to be due to a smaller degree of muscle recruitment related to the smaller loads moved. Nevertheless, in untrained individuals, circuit training with body weight as a load can cause some gains in strength and power, whereas in more endurance trained individuals, this type of training can be less efficient (Waller et al, 2011).

Regardless of strength training type, an adequate volume, frequency and intensity of training needs to be performed in order to stimulate and progressively induce neuromuscular adaptation gains. Training programs should be customized to an individual's specific needs. This generally includes planned progression in intensity and/or volume and appropriate periodization in order to avoid over- or undertraining (Kraemer and Häkkinen, 2002). If the training stimulus in not adequate, detraining would appear. Detraining is characterized by decreases in strength and muscle activation and possible muscle atrophy. The time-course of detraining seems to differ depending on training status, however, typically neural adaptations to strength training decrease before muscle atrophy occurs. The time course of the loss of adaptations tends to mirror the time course of training adaptations. It is importance to note that strength gains may be maintained using a small but adequate volume and intensity of strength training despite an increase in concurrently performed endurance training (Bell et al. 1993).

3.6 Neuromuscular response and adaptations to concurrent strength and endurance training

Many sports require endurance, power, muscular size, and strength at the same time. In order to be successful in such kind of sports, athletes have to practice both strength and

endurance exercises. The inclusion of resistance training (to gain strength, hypertrophy, and power) combined with aerobic exercise in a single program is known as concurrent training.

Interestingly, concurrent training, relative to resistance training alone, has been shown to result in decrements in strength, hypertrophy and power. This phenomenon was firstly discovered by Hickson almost 4 decades ago (Hickson, 1980). Several theories were developed to explain the interference effect. One of the more popular theories is the chronic interference hypothesis, which postulates that the addition of endurance training results in overreaching and overtraining and stimulates competing adaptations over a long-term training program (Leveritt et al. 1999). Further studies in this field allowed for deeper understanding of the phenomenon. Currently, overreaching is thought to be caused by high-volume, high-intensity, or high-frequency training bouts, particularly when bouts of exercise result in large amounts of skeletal muscle damage. It is likely that elements of endurance training, which exacerbate overreaching, would in theory result in greater interference effects (Wilson et al. 2012). The existing data on concurrent training suggests that strength and endurance increase concomitantly up to a point. However, once the frequency increases past 4 days a week or the intensity of endurance exercise increases above 80 % VO2max, endurance exercise prevents the increase in muscle mass and strength that occurs with strength training (Baar, 2014).

Anyway it is worth mentioning that some studies have found little to no decrements in strength training gains with the addition of endurance training (Sillinpää et al 2009, Mikkola et al, 2011). Moreover, recent data have demonstrated large inter-individual variation in responses to changes in maximal voluntary contraction after concurrent training (Wilson et al. 2012). These data indicated that some individuals experience strength decrements after concurrent training, whereas others experience substantial gains. Furthermore, recent studies have also found that the order of exercising modes within one training session (Schumann et al. 2014) as well as the day variations (Eklund et al. 2015) may influence individual's response to concurrent training programme.

The study of Schumann and colleagues (2014) has found an order effect (Endurance + Strength vs. Strength + Endurance) in concentrations of testosterone during 2 days of recovery after the sessions at week 0, which was diminished after 24 weeks of training.

However, the initial difference in testosterone concentrations during recovery did not seem to be associated with strength development, since acute force responses were similar between Endurance + Strength vs. Strength + Endurance. The authors concluded that the loading order of combined training does not seem to influence long-term adaptations of strength development in physically active young men (Schumann et al, 2014).

Most recently, D. Eklund and colleagues (2015) conceived a study that investigated the differences between same day concurrent training with different orders together (SE and ES with different day (DD) concurrent training. After 24 weeks training intervention, it was observed that voluntary activation increased with SE and DD order and that SE had an increase in maximal EMG. The authors speculated that favorable neural adaptations may have been compromised through the long term use of ES order. It was also concluded that a larger training volume, longer period of training and/or training frequency, leads to a more severe neural inhibition, when using the ES order (Eklund et al. 2015).

Taking into account the existing data, it seems that the neuromuscular response to concurrent strength and endurance training will be dependent on the training program content and especially on the intensity and frequency of endurance training. Depending on the content, a combined training program can favor endurance or particular strength type (basic, max, explosive) adaptations. However, an appropriately planned combined training program can improve endurance and/or strength performance depending on the training goal. For example, the study of Rusko and Nummela (2007) has shown that concurrent endurance and explosive type RE improves neuromuscular and anaerobic characteristics in young distance runners. Also both maximal and explosive strength training combined with endurance training have been shown to be more effective in improving strength, power and muscular activation in recreational endurance runners than combined circuit and endurance training (Mikkola et al. 2011). In untrained individuals, combined strength and endurance training improved both neuromuscular and cardiorespiratory fitness with no indication of interference (Sillanpää et al. 2009).

4. CARDIORESPIRATORY RESPONCES TO ENDURANCE TRAINING

The primary functions of the cardiovascular and respiratory systems are to provide the body with oxygen (O2) and nutrients, to rid the body of carbon dioxide (CO2) and metabolic waste products, to maintain body temperature and acid-base balance, and to transport hormones from the endocrine glands to their target organs (Wilmore and Costill, 1994). Those systems are highly responsive to increased activity levels. Low rates of work, such as walking at 4 kilometers per hour place relatively small demands on the cardiovascular and respiratory systems. However, as the rate of muscular work increases, these two systems will eventually reach their maximum capacities and will no longer be able to meet the body's demands.

The cardiovascular system, composed of the heart, blood vessels, and blood, responds predictably to the increased demands of exercise. With few exceptions, the cardiovascular response to exercise is directly proportional to the skeletal muscle oxygen demands for any given rate of work, and oxygen uptake (V ˙O2) increases linearly with increasing rates of work. Cardiac output (Q) is the total volume of blood pumped by the left ventricle of the heart per minute. It is the product of heart rate and stroke volume (SV, volume of blood pumped per beat). The arterial-mixed venous oxygen (A-vO2) difference is the difference between the oxygen content of the arterial and mixed venous blood. A person's maximum oxygen uptake (V˙O2 max) is a function of cardiac output multiplied by the A-vO2 difference. Cardiac output thus plays an important role in meeting the oxygen demands for work. As the rate of work increases, the cardiac output increases in a nearly linear manner to meet the increasing oxygen demand, but only up to the point where it reaches its maximal capacity (Figure 5 A) (McArdle et al. 1994).

Training for endurance capacity generally means training in order to increase cardiac output and to increase capillarization and mitochondrial density. Q is an essential part of maximal oxygen consumption capacity, which is known to be one of the primary determinants of endurance performance. In order to improve oxygen consumption one needs to improve cardiac output by increasing either heart rate (HR) or stroke volume

(Figure 5 B, C) or that one needs to improve arterio-venous oxygen difference (a-vO2diff) by increasing oxygen delivery and/or improving oxygen utilization in the working muscles, the statement above is also known as Fick Principle, and can be presented as equation: VO2 = Q · a-vO2diff (where Q =HR· SV).

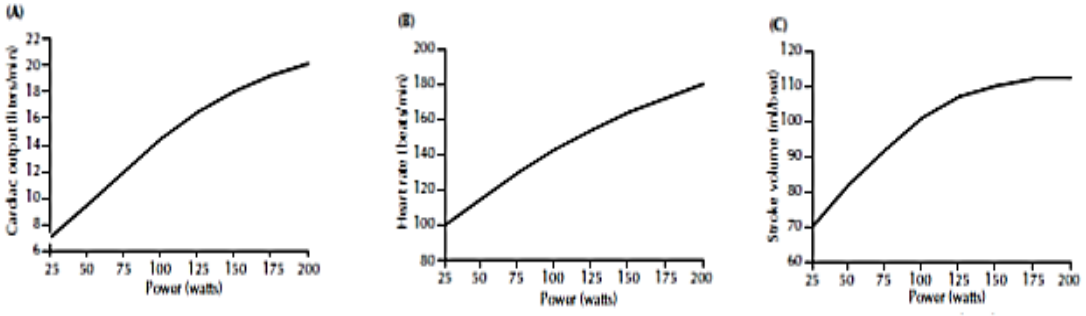

FIGURE 5. Changes in cardiac output (A), heart rate (B), and stroke volume (C) with increasing rates of work on the cycle ergometer (McArdle et al. 1994)

Body's blood flow also increases dramatically during physical activity. At rest, the skin and skeletal muscles receive about 20 percent of the cardiac output. During exercise, more blood is sent to the active skeletal muscles, and more blood is sent to the skin due to increased demand for body temperature control. This process is accomplished both by the increase in cardiac output and by the redistribution of blood flow away from areas of low demand, such as the splanchnic organs (Blomqvist and Saltin, 1983).

The respiratory system also responds when challenged with the stress of exercise. Pulmonary ventilation increases almost immediately, largely through stimulation of the respiratory centers in the brain stem from the motor cortex and through feedback from the proprioceptors in the muscles and joints of the active limbs. During prolonged exercise, or at higher rates of work, increases in CO2 production, hydrogen ions (H+), and body and blood temperatures stimulate further increases in pulmonary ventilation. At low work intensities, the increase in ventilation is mostly the result of increases in tidal volume. At higher intensities, the respiratory rate also increases. Pulmonary ventilation rates can vary from about 10 liters per minute at rest up to 200 liters per minute at maximal rates of work, in highly trained male athletes (Wilmore and Costill, 1994).

All above mentioned cardiorespiratory responses apply to endurance training respectively. The training intensity is the key variable that would drive an acute

response to endurance exercise. Heart rate is the most common and easily accessible cardiorespiratory measure of training intensity as it rises linearly with an increase in training intensity (Figure 5, B). During exercise, failure of the heart rate to increase with increasing workload or a rapid increase in heart rate may indicate significant health problems. In contrast, the lower heart rates observed in athletes are generally the result of increased stroke volume meaning that each beat of the heart is able to pump more blood (Rodeheffer et al, 1984). Although heart rate increases in proportion to workload, the relationship between heart rate and running speed is not constant. In addition, heart rate response to a training session can be influenced by sleep, stress, diet, caffeine consumption, training status and aging.

4.1 Cardiorespiratory adaptations to endurance training

Endurance training leads to significant cardiovascular and respiratory changes at rest and during steady-state exercise at both submaximal and maximal rates of work. The magnitude of these adaptations largely depends on the person's initial fitness level; on mode, intensity, duration, and frequency of exercise; and on the length of training i.e. weeks, months or years (Blomqvist and Saltin, 1983).

According to the principle of training specificity, most pronounced cardiorespiratory adaptations to endurance training include increase of stroke volume and decrease of resting HR. After training, stroke volume is increased at rest, during submaximal exercise, and during maximal exercise; conversely, post-training heart rate is decreased at rest and during submaximal exercise and is usually unchanged at maximal rates of work (Blomqvist and Saltin, 1983). Indeed, the studies have shown that resting heart rate is typically lower in trained individuals; stroke volume is consistently higher in trained athletes than in their sedentary counterparts and it appears to increase systematically until VO2max, suggesting that this cardiorespiratory characteristic develops with endurance training (Weiner & Baggish 2012). The increase in stroke volume appears to be the dominant change and explains most of the changes observed in heart rate and cardiac output.

Several factors contribute to the increase in stroke volume from endurance training. One of them is an increase in blood plasma volume. An increased plasma volume increases

the volume of blood available to return to the right heart and, subsequently, to the left ventricle which in turn leads to the enlargement of left ventricle size (Seals et al, 1994). Another important long term adaptation is an increase in the number of capillaries in trained skeletal muscle, thereby allowing a greater capacity for blood flow in the active muscle. Arterial blood pressure at rest, blood pressure during submaximal exercise, and peak blood pressure all show a slight decline as a result of endurance training in normotensive individuals (McArdle et al. 2009, chapter 15).

Major adaptations in the respiratory system from endurance training are an increase in the maximal rate of pulmonary ventilation, which is the result of increases in both tidal volume and respiration rate, and an increase in pulmonary diffusion at maximal rates of work, primarily due to increases in pulmonary blood flow, particularly to the upper regions of the lung (Wilmore and Costill, 1994).

4.2 Cardiorespiratory response and adaptations to high intensity interval training (HIT)

High-intensity interval training (HIT), in a variety of forms, is today one of the most effective means of improving cardiorespiratory and metabolic function and physical performance of athletes. HIT involves repeated short-to-long bouts of rather high-intensity exercise interspersed by recovery periods. Cardiovascular responses and adaptations observed during typical endurance training also apply to HIT, but with certain features. It has been suggested that HIT protocols that elicit maximal oxygen uptake, or at least a very high percentage of VO2max, maximally stress the oxygen transport and utilization systems and may therefore provide the most effective stimulus for enhancing VO2max (Laursen and Jenkins, 2002). Consequently an optimal stimulus to elicit both maximal cardiovascular and peripheral responses and adaptations is one where athletes spend at least several minutes per session in the zone of at least 90 % of their maximal oxygen uptake (Buchheit and Laursen, 2013). Interestingly, it was suggested that HIT sessions, especially short bout type HIT sessions, might trigger cardiorespiratory adaptations via cardiovascular adjustments occurring specifically during the recovery periods (Fontana et al. 2011).

Several factors determine the desired acute physiological response to an HIT session and the forthcoming adaptations. The intensity and duration of work and relief intervals are the key influencing factors. Then, the number of intervals, the number of series and between-series recovery durations and intensities determine the total work performed. Exercise modality is also of importance when the sport specific adaptations are needed. The manipulation of each variable in isolation likely has a direct impact on metabolic, cardiopulmonary and/or neuromuscular responses and adaptations (Fig. 6).

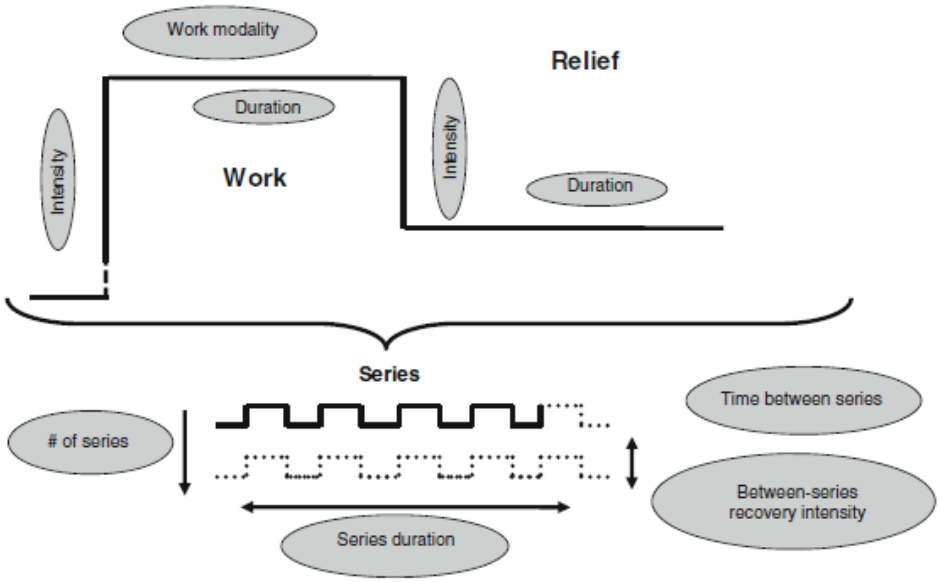

FIGURE 6. Schematic illustration of the nine variables defining a HIT session. (Buchheit and Laursen, 2013)

To conclude, the existing data suggests that HIT sessions, including near-to-maximal long intervals with long recovery durations (i.e. 3–4 min/2 min) might allow athletes to reach a high stroke volume during the work (and possibly the relief intervals). Along these same lines, 4-min intervals at 90–95 % of the speed of VO2max appear to be receiving the greatest interest to improve cardiopulmonary function. Alternatively, repeated short supra-maximal work intervals (e.g. 15–30 s) with long recovery periods (>45 s) might also be effective at reaching high values both during exercise and possibly, in recovery.

4.3 Cardiorespiratory response and adaptations to strength training

The cardiovascular and respiratory responses to episodes of resistance exercise are mostly similar to those associated with endurance exercise. One notable exception is the exaggerated blood pressure response that occurs during resistance exercise. Part of this response can be explained by the fact that resistance exercise usually involves large muscle mass that develops considerable force. Such high, isolated force leads to compression of the smaller arteries and results in substantial increases in total peripheral resistance (Wilmore and Costill, 1994).

The primary focus of strength training is to improve force production characteristics of the muscles. However, strength training also has an effect on cardiorespiratory measures. For example, maximal type strength training causes acute increases in heart rate and oxygen consumption that may be high enough, though short in duration, to induce modest gains in cardiorespiratory fitness when performed over a prolonged period of time. Changes in stroke volume and cardiac output may also be observed with strength training (Weiner & Baggish, 2012). As with endurance training, current training status may influence individual's cardiorespiratory responses and adaptations to strength training.

Regarding cardiovascular adaptations, circuit strength training can differ from traditional strength training. Circuit type strength training performed with modest / high intensities has been reported to cause modest gains in aerobic capacity because of the heart rate response that can reach approximately 80% of maximum heart rate (Klika and Jordan, 2013). As well as with endurance training, the intensity of the exercises is the primary factor that drives the cardiovascular adaptations.

4.4 Cardiorespiratory response and adaptations to concurrent strength and endurance training

An acute cardiorespiratory response can greatly vary depending on the exercise mode, intensity and duration, as well as substrate availability and utilization capacity. Although there is some evidence that metabolism may be altered, at least immediately

post training session, depending on the order of combined exercises performed (Taipale et at, Schumann et al. 2014).

Combined strength and endurance training does not appear to induce large increases in VO2max, when the subjects are already physically active or elite (Millet et al. 2003; Mikkola et al. 2007). One explanation behind this lack of improvement may be an insufficient volume and intensity of the endurance training involved compared to the normal endurance training programs, or may be a result of combined strength and endurance training study designs often replacing some percentage of endurance training with strength training.

Despite the evidence suggesting that combined strength and endurance training actually helps to improve parameters of endurance performance, improvements in aerobic capacity may actually be blunted by strength training when the interference effect occurs (Hickson, 1980, Glowacki et al. 2004). However, a number of studies have reported that endurance capacity or its development is not interfered by strength training (Paavolainen et al. 1999; Millet et al. 2002; Mikkola et al. 2007; Wilson et al. 2012).

In appropriately planned combined training (i.e. when no interference effect is present), cardiorespiratory responses to a single session of combined strength and endurance training as well as long term adaptations to combined training period may be expected to produce similar responses as for single mode training session/period (see parts 4.0 – 4.3). Nevertheless, the prospect that resistance training can actually decrease capillary and mitochondrial volume density or increase muscle mass, which in turn will negatively affect endurance performance, may deter endurance runners from incorporating strength training into their training program. Therefore, planning of such sessions should be done with caution to prevent any maladaptation.

5. METHODS

5.1 Subjects

A total of 9 subjects volunteered to participate in the present study. Average age, body mass, and height were 30.7 years (± 5.8), 76 kg (± 5), 178.3 cm (± 5.7). An average fat percent was 12.7 (± 4.5). Those 9 subjects completed all study requirements and were considered in analyses. All subjects were healthy and physically active, participating in endurance type exercise, such as jogging, cycling, and cross-country skiing. All subjects were recruited through advertisement and email lists. The subjects gave their written informed consent to participate in the study. Subjects received full explanation of the details and rationale of the study and were informed that they were free to withdraw at any time. The possibility that electrical stimulation might cause discomfort was fully explained as well as the nature of the risks involved. The study was approved by the ethical committee of the University of Jyväskylä.

5.2 Experimental Design

The subjects made 6 visits to the laboratory for the neuromuscular (NM) measurements and one visit for the familiarization session during the control period. During the familiarization visit, the subjects were accustomed to neuromuscular function assessment and performed a unilateral knee extension with electrical stimulation of quadriceps muscles. The familiarization session and the Pre-test were separated by 3-5 days. Additional two visits (pre and post intervention) were made to perform the maximal oxygen consumption running test on the treadmill. Another three visits to the laboratory were made in the morning hours on weeks -1, 4 and post 8 to perform 1 repetition maximum (RM) leg press. On the same day evening the subjects performed 3000m time trail using indoor athletic track. Pre-tests and Mid-tests were separated by 4 weeks, as well as, Mid-test and Post-test. The experimental design used in the study is summarized in Figure 7.

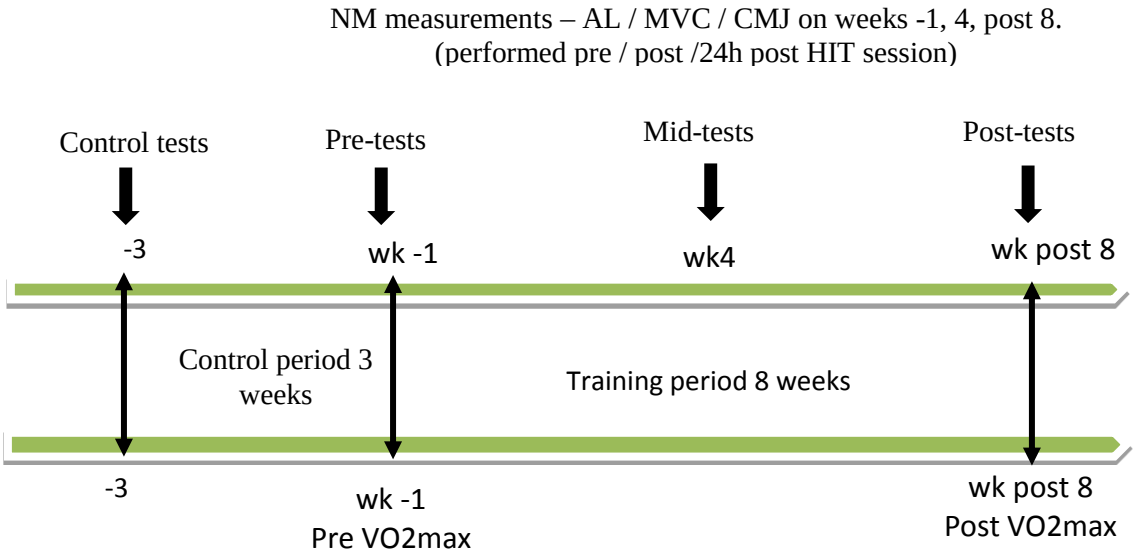

FIGURE 7. Experimental Design

5.3 Training Programme

Before the start of the training programme the subject performed a 3 week control period. During those weeks subjects were allowed to continue their regular training, meaning 6-8h of total weekly training volume. One high intensity interval training session (3x10x30s protocol) and one strength training session were predefined during each week of the control period to familiarize the subjects with these sessions before the training period. The rest of the training hours were low intensity endurance training under aerobic threshold. Time duration of the training sessions was limited by 60 min. It was also recommended to have one rest day a week.

The actual training period of the study consisted of 8 weeks. Those 8 weeks were comprised of 2 similar periods of 4 weeks each. The structure of one 4 week period is presented in Figure 8. The detailed programme of each week of 4 weeks training period is presented in Table 2.

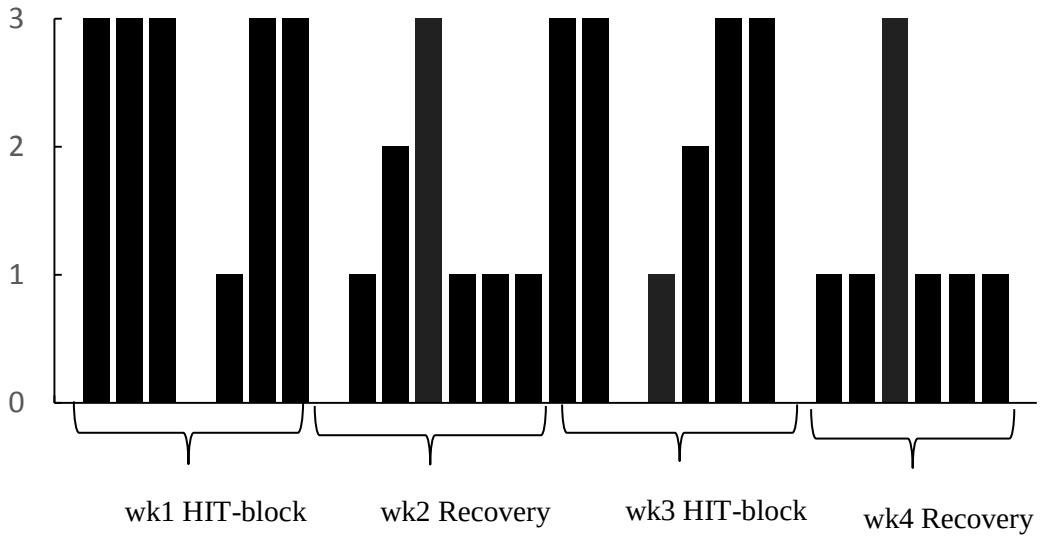

FIGURE 8. The Structure of 4 week training period; 0=Rest; 1=low intensity training; 2=Strength training; 3=High intensity interval training

TABLE 2. A detailed plan of the 4-week training period.

DAY #	Week 1	Week 2	Week 3	Week 4
1	3000m TT	REST	HIT 4 x 4 min	REST
2	HIT 4 x 4 min	LIT	HIT 3 x10x30sec	LIT
3	HIT 3 x 10 x 30sec	ST	REST	LIT
4	REST	HIT 4 x 4 min	LIT	HIT 3 x10x30sec
5	LIT	LIT	ST	LIT
6	HIT 4 x 4 min	LIT	HIT 3 x10x30sec	LIT
7	HIT 3 x 10 x 30sec	LIT	HIT 4 x 4 min	LIT

TT- time trail; HIT – high intensity interval training; LIT – low intensity interval training; ST – strength training.

The intensity for HIT sessions was calculated individually for each subject based on the results of the treadmill VO2max test performed during the control period. For the 4x4min HIT protocol the target intensity was set at 90-95 % of vVO2max, for 3x10x30s HIT protocol the target intensity was 95 % of vVO2max. The 3 x 10 x 30s HIT protocol was also used as a loading mode in the neuromuscular tests on weeks -1, 4 and post 8, the intensity was set at 95% of vVO2max.

Strength training session comprised of 7 exercises: 1) dynamic leg press 2 x 8-10 x 75-85 % 1RM; 2) body weight unilateral box step ups (box height 30cm) 3 x 5; 3) squats 3 x 6 x 30/40/60 % 1RM, 4) knee flexion using machine 2 x 10 x 60-70 % 1RM; 5) 3 x 20 Swiss knife exercise for abdominal muscles; 6) bench press 3 x 8-10 x 70-75 % 1RM; 7) Lat pull down 3 x 8-10 x 70-75 % 1RM. Two minutes were allowed for recovery between sets.

5.4 Data Collection

On the neuromuscular measurement days the subjects performed maximal unilateral isometric knee extension with electrical stimulation of quadriceps muscles and counter movement jump (CMJ) tests. After the completion of the tests the subjects performed a high intensity interval training session on the treadmill (3 x 10 x 30sec protocol). The speed was individually adjusted for each subject according to 95% of their speed at VO2max. During the session the subject's heart rate (HR) was constantly recorded and blood lactate samples from the fingertip were taken pre and post HIT session. Immediately after the cessation of HIT session CMJ and isometric knee extension measurements were repeated. After 24h from the end point of the measurements the subjects returned to the laboratory to perform maximal unilateral isometric knee extension with electrical stimulation of quadriceps muscles and counter movement jump (CMJ).

On 1RM leg press and 3000meters measurement days the subjects performed dynamic 1RM leg press in the morning, after that, at least 5h were given for recovery. After the recovery period 3000m running time trail was performed. The detailed description of the testing process is presented in the following parts of the thesis.

5.4.1 Anthropometric measurements

Body mass was measured by a calibrated scale before each round of the measurements i.e. on weeks -1, 4 and post 8 after a 12 hour, overnight fast. Height was measured by a wall mounted scale. Whole body fat percentage and total muscle mass was measured by an eight-polar bioelectrical impedance device (InBody 720 body composition analyzer, Biospace Co. Ltd, South Korea). The subjects were upright with the arms abducted by approx. 20° to ensure that the arms and trunk were not in contact.

5.4.2 Isometric knee extension

A modified David 200 knee extension device (David Health Solutions Ltd, Helsinki, Finland), with locking system and strain gauges (Häkkinen et al. 1987), allowed assessment of maximal unilateral isometric knee extension. Subjects were secured by a non-elastic strap at the hip and a pad across the knee to prevent extraneous movement with a knee angle of 107° and hip angle of 110° (Figure 9). The subjects were instructed to perform 3 maximal isometric contractions by gradually increasing force over a 3–5 s period. This was performed pre HIT, post HIT, and 24h post HIT session on weeks: -1, 4 and post 8 respectively.

A superimposed twitch was evoked at the peak force to assess voluntary activation in all of the test repetitions. Maximal unilateral isometric force (sampled at 2000 Hz, and filtered by a 20 Hz low-pass 4th order Butterworth filter) was considered as the greatest voluntary evoked force prior to the superimposed twitch. Verbal encouragement and visual feedback was provided during all contractions.

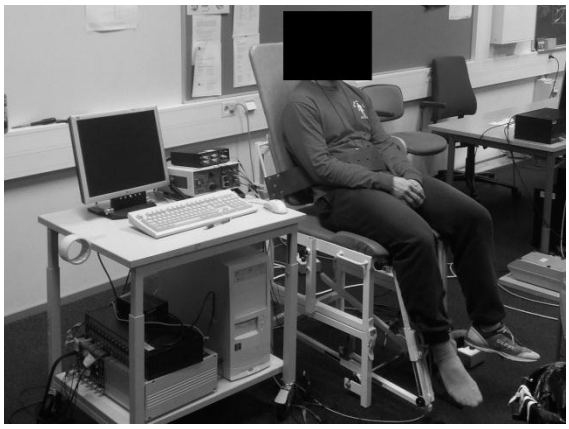

FIGURE 9. Subjects position for Unilateral Knee Extension

5.4.3 Muscle electrical stimulation

Muscle stimulation was performed by placing four, galvanically paired, self-adhesive electrodes (6.98 cm V-trodes, Mettler Electronics Corp, USA) on the proximal and mid regions of the quadriceps muscle belly (Figure 10). Single 1ms rectangular pulses were delivered by a constant-current stimulator (Model DS7AH, Digitimer Ltd, UK) until a

torque plateau was observed. An additional 25 % of stimulation current was added to the current identified to produce maximum torque.

During unilateral maximum isometric knee extension trials, the same single pulse stimulation was delivered during the plateau of peak torque and then one more pulse 2 sec after contraction cessation to assess voluntary activation (Merton 1954). Voluntary activation was assessed from the additional torque produced by the superimposed twitch and the maximum torque of the subsequent resting twitch using the formula of Bellemare and Bigland-Ritchie (1984); activation % = [1 – (Pts/Pt)] × 100. According to the study design these measurements were performed pre, post and post 24h post HIT session on weeks: -1, 4 and post 8 respectively.

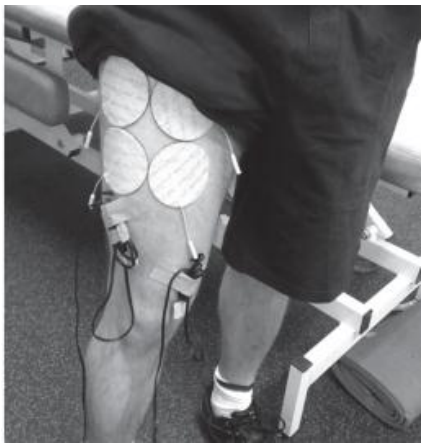

FIGURE 10. The placement of Stimulation Electrodes

5.4.4 Counter Movement Jump

A force platform (Department of Biology of Physical Activity, Jyväskylä, Finland) was used to measure maximal dynamic explosive force by countermovement jump height. Subjects were instructed to stand with their feet approximately hip-width apart with their hands on their hips (Figure 11). The countermovement jump action was then demonstrated and explained to be quick and explosive so that knee angle for the jump was no less than 90 degrees. Force data was collected and analyzed by computer software (Signal 2.14, CED, Cambridge, UK), which used the equation h = I*2 x 2gm*2 to calculate jump height from impulse (I = impulse, g = gravity and m = mass of subject).

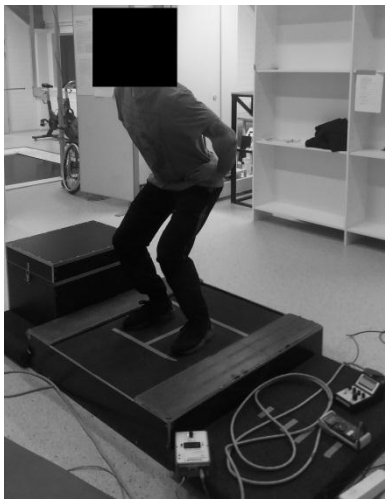

FIGURE 11. Counter Movement Jump test using the force plate

5.4.5 Dynamic Leg press

Maximal bilateral concentric one repetition maximum (1RM) was measured during the leg press. Prior to attempting 1RM, the subjects completed a warm-up, after which no more than 5 attempts to reach 1RM were made. The knee angle at the beginning of the leg extension was approximately 60 degrees. Subjects were instructed to grasp handles located by the seat of the dynamometer and to keep constant contact with the seat and backrest during leg extension to a full 180 degrees (Figure 12). Thereafter, single repetitions using 5 kg increments were performed until the subject could no longer lift the load to full extension. The greatest weight that the subject could successfully lift (knees fully extended) was accepted as 1RM.

FIGURE 12. Dynamic Leg Press test

5.4.6 Cardiorespiratory performance by the VO2max test

Maximal aerobic capacity was measured by maximal oxygen uptake (VO2max) using a treadmill running protocol (Mikkola et al. 2007). Running velocity began at 8 km· h-1. Running velocity was increased by 1 km· h-1 every third minute until volitional exhaustion. Treadmill incline remained a constant 0.5 degrees. Heart rate was recorded continuously using a heart rate monitor (Garmin XT920, Garmin Ltd, Schaffhausen, Switzerland). Oxygen consumption was measured breath-by-breath throughout the test using a gas analyzer (OxyconPro, Jaeger, Hoechberg, Germany) and VO2max was accepted as the highest average 60 s VO2 value. For blood lactate sampling, fingertip samples were taken every 3 min while the treadmill was stopped for approximately 15-20 s. The results of VO2 max tests are beyond the scope of this thesis so they will not be presented.

5.4.7 Cardiorespiratory performance by 3000 meter time trail

3000 meter running time trail was performed in indoor athletics track (HipposHalli, Jyvaskyla, Finland). 200 meter laps were used for the test. Intermediate times were taken after every kilometer for each subject. Time trials were run in groups of an average 4. The groups remained the same through the study. Verbal encouragements were provided for the subject along the time trail.

5.4.8 Warm up protocols

Prior to pre-HIT and 24h post – HIT neuromuscular measurements the subjects completed a 10min warm up including 7min of stationary bike cycling with intensity under anaerobic threshold and 3 min of dynamic full body stretching. Prior to HIT sessions the subjects completed a 10min running warm up on the treadmill at the speed of Aerobic Threshold including two 15sec – 20sec accelerations with the speed of 95% of vVO2max performed at min 5 and min 8 respectively.

5.5 Data analysis

Standard statistical methods were used for calculation of means and standard deviations

(SD). Within group differences were analyzed using repeated measures factorial ANOVA. The criterion for significance was $p < 0.05$. Statistical analyses were completed with SPSSWIN 14.0 - 18.0 (SPSS Inc., Chicago, IL, USA).

6. RESULTS

6.1 Changes in Neuromuscular variables during 8 weeks of high intensity interval training

The changes in pre / post / 24h post HIT session Activation Level (AL) percent during 8 weeks of high intensity interval training are presented in Figure 13. A significant decrease of AL by 8.2 % ± 7.9 (from 81.4 ± 7.0 in pre HIT to 75.7 ± 9.6 post HIT) was observed only during the mid-measurements (week 4). A significant difference 7.1 % (± 5.9) between post HIT and 24h post HIT AL was observed only during the week 4 measurements too. No significant differences were observed between pre HIT and 24h post HIT AL on weeks -1, 4 and post 8, respectively. No significant difference was observed between pre HIT AL on week -1 (80. 6) and pre HIT AL on week post 8.

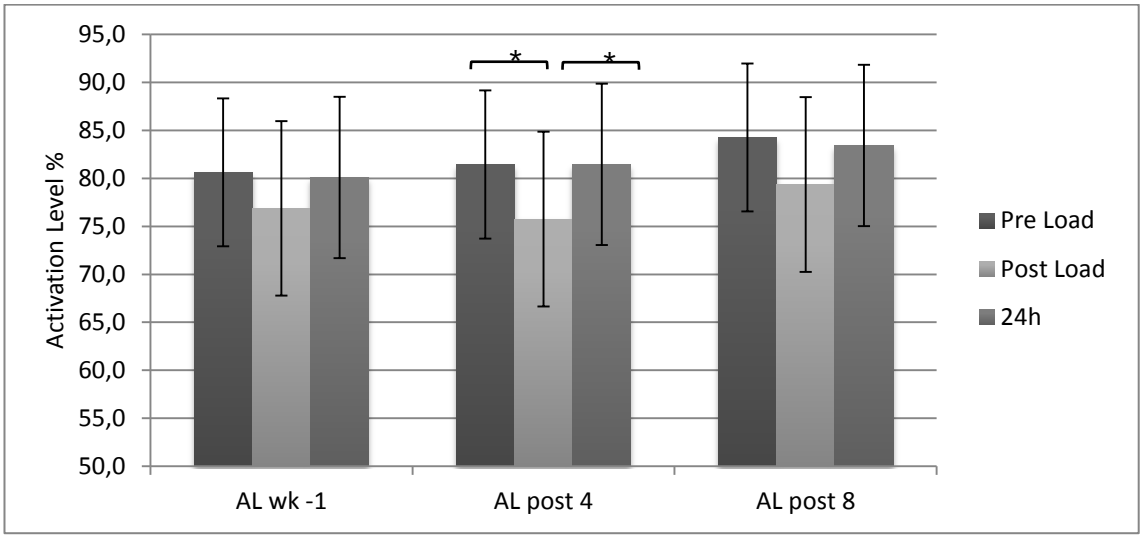

FIGURE 13. Changes in pre / post / 24h post HIT session AL during 8 weeks of high intensity interval training. Significance set at: * = $p < 0.05$

6.1.1 Changes in Maximal Voluntary Contraction Force (MVC) after HIT session and 24 hours of recovery

The changes in pre / post / 24h post HIT session MVC force during 8 weeks of high intensity interval training are presented in Figure 14. A significant decrease from pre to post HIT maximal voluntary force was observed only during the mid-measurements (week 4), being 12.3 % ± 8.6 (from 684.7 N to 614.1 N). No significant differences were observed between pre HIT and 24h post HIT MVC force and between post HIT and 24h post HIT MVC force on weeks -1, 4 and post 8. No significant differences

were observed between pre HIT MVC force on weeks -1 (651.4 N) and pre HIT MVC force on weeks 4 and post 8, respectively.

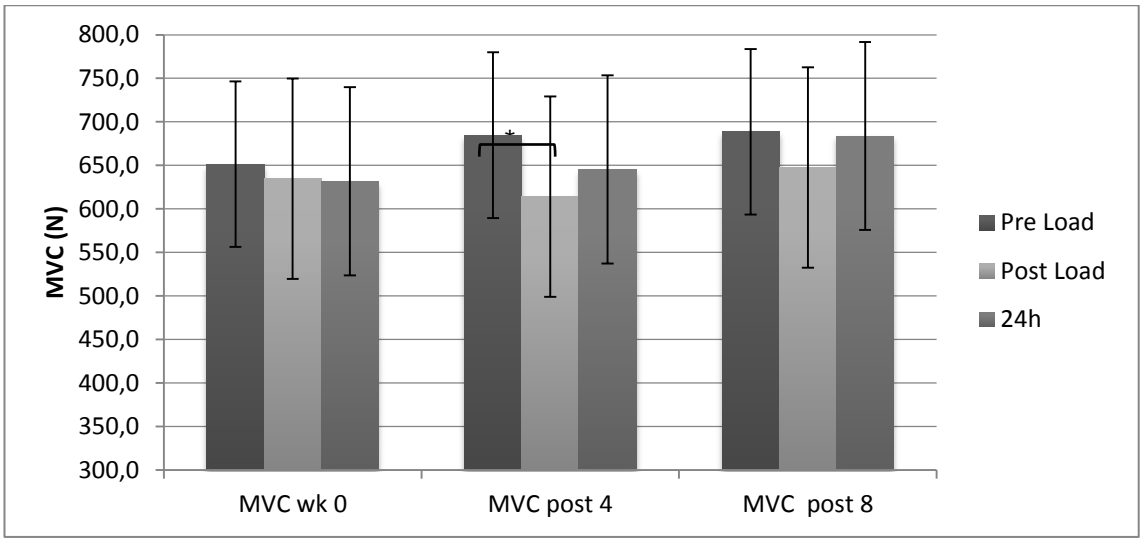

FIGURE 14. Changes in pre / post / 24h post HIT session MVC force during 8 weeks of high intensity interval training. Significance set at: * = $p < 0.05$

6.1.2 Changes in Counter Movement Jump (CMJ) height after HIT session and 24 hours of recovery

The changes in pre / post / 24h post HIT session CMJ height during 8 weeks of high intensity interval training are presented in Figure 15. A significant increase from pre to post HIT jumping performance was observed only during the pre-measurements on week -1, being 8.0 % ± 6.3 (from 26.7 cm to 29.2 cm); and the mid-measurements on week 4, being 7.7 % ± 6.8 (from 26.9 cm to 29.4 cm) but not on week post 8. Blood lactate values in the mid and post-tests were 5.7 mmol/L ± 1.4; pre-test 6.5 mmol/L ± 2.1. A significant decrease was observed from post HIT to 24h post HIT CMJ height on week -1 (8.4 % ± 8.7) and week 4 (11.6% ± 8.4), but not on week post 8. No significant differences were observed between pre HIT and 24h post HIT jumping height on weeks -1, 4 and post 8 respectively.

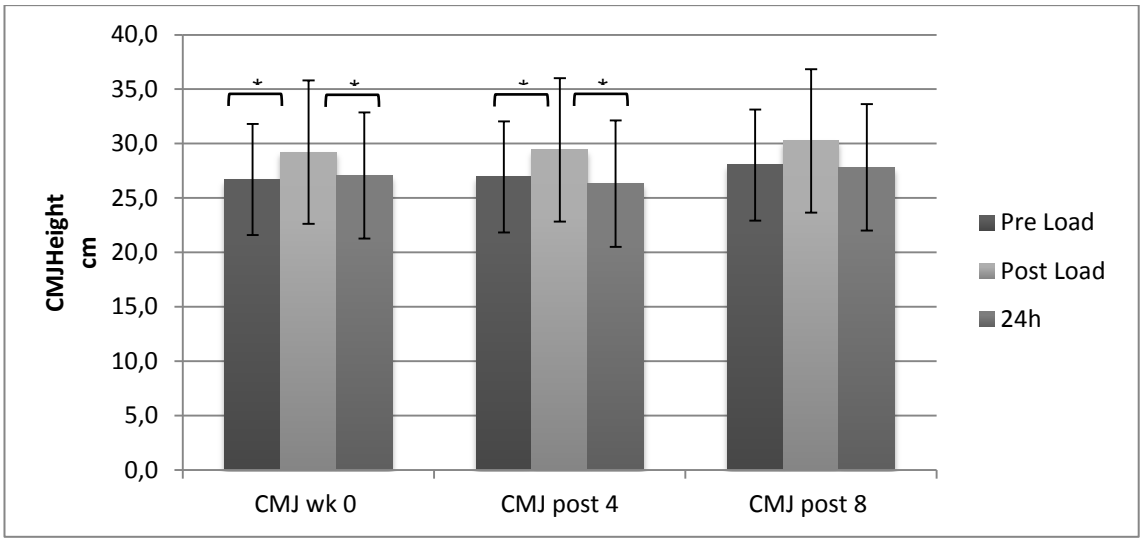

FIGURE 15. Changes in pre / post / 24h post HIT session CMJ height during 8 weeks of high intensity interval training. Significance set at: * = $p < 0.05$

6.2 Changes in one repetition maximum (1RM) dynamic leg press

The changes in 1RM leg press during 8 weeks of high intensity interval training are presented in Figure 16. 1RM performance progressively increased from week -1 to week post 8, while a significant increase was observed only from week 4 to week post 8 (8.4 % ± 4.9) and from week -1 to week post 8 (8.9 % ± 4.9). No significant difference was observed between 1RM values from week -1 to week 4.

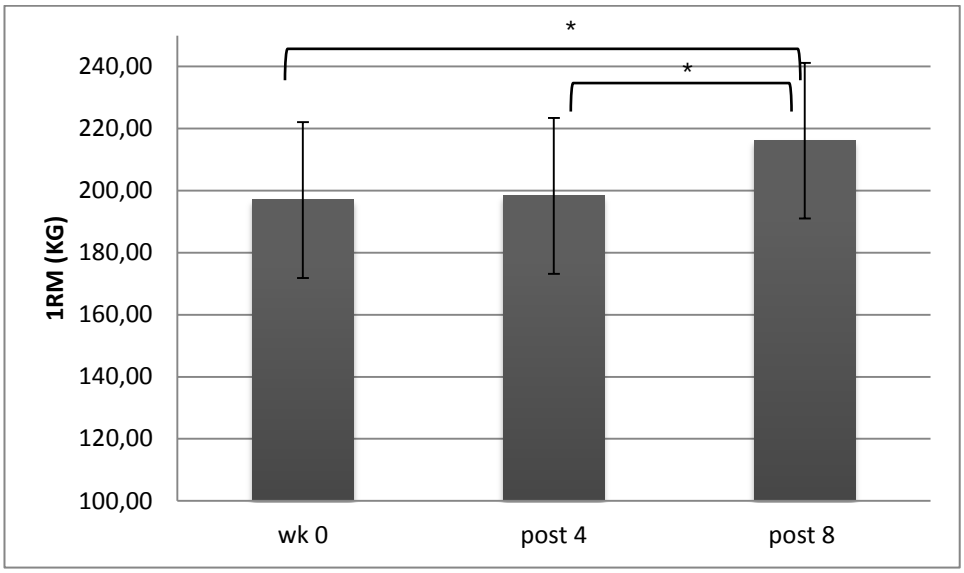

FIGURE 16. Changes in one repetition maximum dynamic leg press during 8 weeks of high intensity interval training. Significance set at: * = $p < 0.05$

6.3 Changes in 3000 meters Time Trail (TT)

The changes in 3000 meters TT performance during 8 weeks of high intensity interval training are presented in Figure 17. 3000m times progressively decreased from week -1 (10:47 ± 00:52:80) to week post 8 (10:16 ± 00:49:14). Significant improvements were observed between all the time points, being: 3.4% ± 1.9 from week -1 to week 4, 1.5 % ± 0.8 from week 4 to week post 8 and 4.9 % ± 2.0 from week -1 to week post 8 respectively.

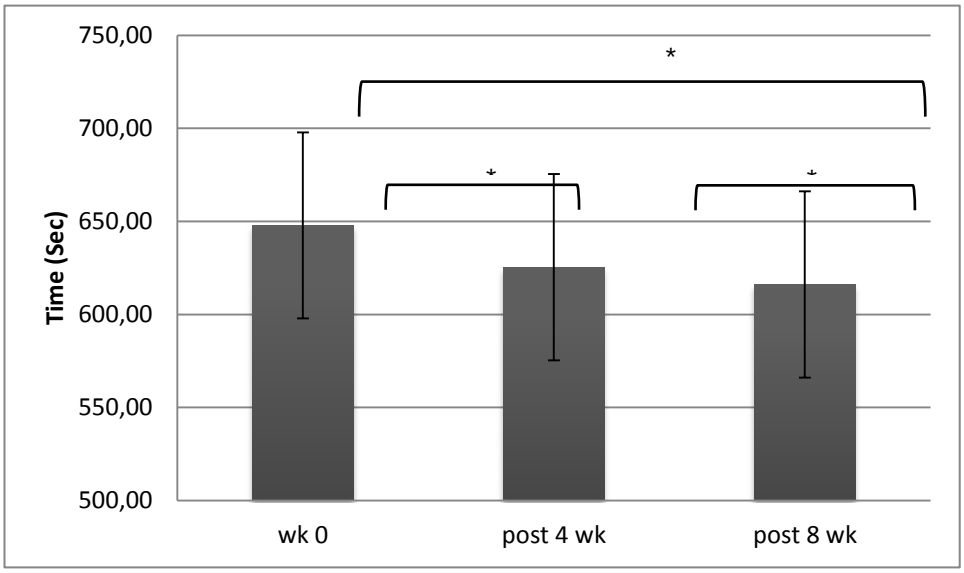

FIGURE 17. Changes in 3000 meters TT performance during 8 weeks of high intensity interval training. Significance set at: * = $p < 0.05$

6.4 Correlations between relative changes in Neuromuscular and Aerobic variables

There were no significant correlations between changes in AL percent from pre HIT to post HIT and changes in MVC force from pre HIT to post HIT on week -1, week 4 and week post 8. A positive significant correlation was observed from week -1 to week post 8 between changes in Pre HIT AL percent and Pre HIT MVC force ($r = 0.790$; $p = 0.011$).

No significant correlations were observed between changes in AL percent and MVC force from pre HIT to post HIT and changes in CMJ height from pre HIT to post HIT on weeks: -1, 4 and post 8. No significant correlations were found between relative

changes in pre / post / 24h post HIT CMJ height and relative changes in pre / post / 24h post HIT MVC force and AL from week -1 to week post 8.

No significant correlations were found between week -1 to week post 8 relative changes in 1RM leg press and pre HIT, AL, MVC and CMJ. Positive but not significant correlations were observed between changes in 1RM leg press and changes in 3000m times from week -1 to week post 8. Also positive but not significant correlations were observed between changes in 3000m times and changes in pre HIT values for: AL, MVC, CMJ from week -1 to week post 8.

7. DISCUSSION

The purpose of the present study was to investigate the acute HIT session induced changes and recovery in muscle activation level, voluntary force production and counter movement jump performance during 8 weeks of HIT training in recreationally endurance trained men.

The main findings of the present study were:

1. There was a significant decrease in MVC force and muscle AL percent from pre to post HIT session on week 4, while on weeks -1 and post 8 the decrease was not significant.

2. There was a significant improvement in CMJ height from pre to post high intensity interval session on weeks -1 and 4, but not on week post 8.

3. 24 hours seemed to be enough to recover AL, MVC and CMJ capacities back to pre-HIT levels in recreationally endurance trained men, since no significant changes were observed between Pre HIT and 24h post HIT values on weeks -1, 4 and post 8.

4. A significant increase in 1RM leg press performance was observed after 8 weeks of high intensity interval training with 0.6 strength training sessions per week.

5. A significant improvement in 3000 meters time trail performance was observed after 8 weeks of high intensity interval training.

6. No significant correlations were found between performance changes in neuromuscular, strength and endurance measurements.

7.1 Acute changes in neuromuscular variables after HIT session

In the present study an acute decrease in subjects' muscle AL and MVC force took place immediately after HIT session, while a significant decrease was observed only during the mid-measurements on week 4. Endurance training is generally known to produce a smaller magnitude of neuromuscular fatigue compared to strength training (Paavolainen et al. 1999). However, this does not mean that the levels of fatigue after endurance training are always low. It has been previously shown that both neuromuscular and peripheral fatigue increase with increasing intensity of the exercise (Froyd et al. 2016).

A significant neuromuscular fatigue was observed only at the mid measurements (week 4). Taking in account that the subjects were given 3 days of low intensity training (LIT) before every time point of the measurements (weeks -1, 4 and post 8) the subjects were expected to be in the similar conditions before pre, mid and post measurements.

It can be speculated that if those 3 LIT sessions were completed with higher intensity and/or longer duration compared to the pre-test period, this might lead to lack of recovery from previous HIT sessions, which in turn would lead to the summation and accumulation of fatigue from the previous sessions. The phenomenon of summative and residual fatigue is a well-known fact, which can be especially significant during a block periodization model, when high load is more concentrated than during a traditional periodization model (Rønnestad et al, 2012; Issurin, 2008, 2010). However, the subjects did not report any sign of overtraining before the mid-measurements and the RPE values reported by the subjects during the HIT session did not differ from the pre-test. In addition, mean lactate values at the end point of the session did not differ from the post-test (5.7 vs 5.7 mmols/L) but were lower than in the pre-test (5.7 vs 6,5 mmols/L). This supports the fact that the stress from the mid test HIT session was not higher compared to the other time points of the measurements.

The observed significant decreases in AL percent (8.2 ± 7.9) and MVC (12.3 % ± 8.6) at week 4 are in line with the existing literature. For example, Fernandes et al (2013)

reported that the decrement scores from 5 to 20 % is generally observed following HIT sessions, suggesting large post HIT neuromuscular function impairment. However, it is important to remember that there could be great individual differences in subjects' response to HIT session due to individual training status and specificity of the HIT protocol (Buchheit and Laursen, 2013). This also applies to the current study as high standard deviations among the subjects were observed. The variations in subjects post HIT fatigue levels could be explained by the differences in aerobic and fatigue resistance capacities i.e percent of oxidative fibers, mitochondrial content and activity, muscle capillarization and stroke volume (Buchheit and Laursen, 2013).

The absence of significant post HIT fatigue on week post 8 might be a result of subjects' adaptations to training. This assumption is supported by the lower end session blood lactate values compared to the pre-test (5.7 ± 1.4 vs 6.5 ± 2.1) and lower mean RPE values reported during the HIT session. It has been previously shown, that aerobic capacity can significantly improve after 8 weeks of training, especially when HIT training protocols are used (Laursen and Jenkins, 2002; McArdle et al, 2009; Buchheit and Laursen, 2013) and therefore, by week post 8 the sessions were not such fatiguing due to better fatigue resistance capacity of the subjects.

The fact that both AL and MVC decreased indicates that both central and peripheral acute fatigue was present following the HIT session. While peripheral fatigue indicates the alterations to muscle excitability and excitation-contraction coupling, related to intramuscular potassium concentration disturbance and accumulation of metabolic by-products, the presence of significant central fatigue shows that the muscle recruitment capacity of the central nervous system was impaired too (Gandevia, 2001). The results from week 4 showed that the relative decrease in MVC was higher than the relative decrease in AL, meaning that the acute neuromuscular fatigue was more peripheral in origin. This finding is in line with the current literature. For example, Thomas et al (2015) observed greater levels of central fatigue after longer distance cycling (40 & 20km) but a higher level of peripheral fatigue after shorter and more intensive bouts of exercise (4 km). The predominance of peripheral fatigue during short bout HIT sessions was also shown by Perrey et al. (2010). The authors suggested that fatigue induced by HIT including either very short (<20 s) to short (<1 min) and/or non-maximal efforts (<120 % of v VO2max) tends to be predominantly peripheral in origin. The HIT

protocol used in the current study (3 x 10 x 30 sec at 95 % v VO2max) fits to those parameters. It is also worth mentioning that central fatigue is predominating after the HIT protocols that include repeated long (>30 s) and supra maximal all-out sprints (Fernandez et al. 2013), which were not used in the present study.

No significant correlations were found between changes in pre to post HIT AL and MVC as well as between AL & CMJ and MVC & CMJ during 8 weeks of high intensity interval training. The absence of the significant positive correlation between MVC/AL and CMJ was clearly shown by acute post HIT changes in AL, MVC and CMJ. Jumping performance improved, while AL and MVC decreased. This finding is in line with the results of Boullosa et al. (2011) who measured concurrent decrease in maximum force capacity but observed improved CMJ ability after the maximum running speed test. King et al. (2013) did not find any significant correlations between lower extremity fatigue and changes in jumping performance which supports the findings of the present study. In the present study, post HIT neuromuscular fatigue was not elevated to a sufficient level to decrease jumping performance but it was sufficient to impair isometric force production capacity and muscle activation levels.

The observed significant increase in CMJ performance following the HIT session on weeks -1 and 4, indicates the presence of post exercise potentiation. Interestingly, the same phenomenon was observed by Vuorimaa et al (2006) and Boullosa et al. (2009, 2011) who have shown that trained middle and long distance runners who completed intensive running have increased their jump performance. Even though the loading modes in the present study and the studies of Vuorimaa et al and Boullosa et al were different, post activation potentiation (PAP) in CMJ performance was still observed, indicating that it might be more dependent on the endurance background of the subjects rather than on the loading mode.

Post activation potentiation (PAP) refers to the phenomenon by which neuromuscular performance characteristics are acutely enhanced as a result of their contractile history (Boullosa et al. 2011). Physiological mechanisms of the PAP phenomenon remain to be not fully explained. It was suggested by the previous studies that increased CMJ height could be the result of improved utilization of elastic energy (Vuorimaa et al, 2006). This phenomenon might allow the muscle fibers to reuse more elastic energy stored during

the stretch-shortening cycle. It the current study it could be assumed that improved CMJ height results from enhanced capacity to use stored elastic energy that counteracted the force loses. Those finding are in line with the results of the previous studies in this field (Vuorimaa et al 2006, Boullosa et al 2009, 2011).

7.2 Neuromuscular recovery patterns at 24 hours post HIT session

The present study showed that 24 hours seemed to be enough to lead to recovery in AL, MVC and CMJ capacities back to the pre-HIT levels in recreationally endurance trained men, since no significant differences were observed between pre HIT and 24h post HIT values at any time points of the measurements (week -1, 4 and post 8). The findings of the present study are in line with the existing data, which indicates that it generally takes about 48 h to recover neuromuscular performance capacity after the HIT session (Buchheit and Laursen, 2013). However, it is important to keep in mind that the existing data on neuromuscular fatigue following endurance and HIT protocols is still limited and many training protocols still require investigation. The existing data shows that the recovery patterns can vary from few hours up to 2 – 5 days following training, due to variations in exercise protocols and training status of the athletes (Sherman et al. 1985; Gomes et al. 2002; Millet et al. 2004; Froyd et al. 2013; Wiewelhove et al. 2015). It is important to remember that responses to HIIT and levels of fatigue as well as recovery patterns appear to be highly individual.

It can be speculated that the absence of significant post 24h neuromuscular fatigue is most likely caused by the absence of significant post HIT session muscle damage and inflammation. Since no data on CK or other markers of muscle damage is available, it is not possible to ensure that no muscle damage / inflammation were present. It has been clearly shown that when such factors as muscle damage and inflammation occur, the neuromuscular performance can be impaired up to 3-5 days (Sherman et al. 1985; Gomes et al. 2002; Wiewelhove et al. 2015).

7.3 Changes in strength and endurance performance after 8 weeks of HIT training

The present study showed that even 5 strength training sessions performed during 8 weeks when combined with HIT training can lead to significant improvements in

strength test performance (1RM leg press). Interestingly, no significant improvements were observed after the first 4 weeks of training, while a significant increase in strength performance took place from week 4 to week post 8. The absence of significant improvements after 4 weeks of training could occur due to insufficient training volume and or frequency for adaptations. The existing literature shows that it generally takes from 4 to 12 weeks of training to obtain early-phase adaptations (Häkkinen et al. 1985a; Häkkinen et al. 1985b).

The early-phase adaptations are mainly neuromuscular in origin i.e synchronization and improved recruitment of additional motor units (Häkkinen, 1994). It can be suggested that early-phase neuromuscular adaptations explain the improvement in 1RM dynamic leg press performance in the present study, since no significant difference in subjects' lean muscle mass was observed from pre to post intervention measurements. This is also in line with the existing literature demonstrating that hypertrophy usually occurs as a long term adaptation after prolonged training (Häkkinen, 1994). It should be noted that the frequency of strength training was very low in the present study, while the amount of HIT sessions was high. Taking that into account, it could be suggested that HIT sessions when performed with sufficient frequency and duration can provide enough stress to awoke early phase neuromuscular adaptations patterns as for strength training.

One step further from this study would be to investigate how an increase in strength training frequency would influence strength and endurance parameters of the athletes. It is well accepted that strength and endurance can increase concomitantly up to a point, but after that point interference effect may occur in maximal strength (Hickson, 1980; Leveritt et al. 1999; Baar, 2014) and even more in explosive strength (Häkkinen et al. 2003). The present investigation has shown that low frequency strength training (0.6 a week) combined with high frequency HIT (3-5 a week) training does not lead to interference effect as both strength and endurance parameters were continuously increasing following 8 weeks of training.

As was mentioned before, endurance performance (i.e 3000m Time Trail) was continuously improving during 8 weeks of HIT training. This finding is not novel and many investigators have shown that high intensity interval training is an effective tool for improving endurance performance in recreational and even professional athletes

(Helgerud et al 2007; Daussin, 2008; Seiler and Tonnessen, 2009; Stoggl and Sperlich, 2014). Primary mechanisms that stand behind the benefits of HIT training, is that such sessions when performed at the speed of maximal oxygen uptake, or at least a very high percentage of VO2max maximally stress the oxygen transport and utilization systems and may therefore provide the most effective stimulus for enhancing VO2max and endurance performance (Laursen and Jenkins, 2002). It was also shown that optimal stimulus to elicit both maximal cardiovascular and peripheral responses and adaptations is one where athletes spend at least several minutes per session in the zone of at least 90 % of their maximal oxygen uptake (Buchheit and Laursen, 2013). The HIT protocols used in the present study allowed athletes to achieve few minutes in the zone of at least 90 % of their maximal oxygen uptake.

Interestingly, even both running and maximal bilateral 1RM leg press performance improved significantly, no significant correlations were found between changes in those variables. It has been shown by several studies that strength and running performance can improve concomitantly but the improvements are not necessarily correlated (Storen et al. 2008; Ferrauti et al 2010; Vikmoen et al 2016). Hypertrophic type of strength training does not put enough stress to the cardiovascular system to achieve significant cardiovascular adaptations, so the gains from the weight room might not be transferred into improved running performance. In the present study the primary improvements in 3000m TT were achieved by the adaptation caused by high intensity interval training.

7.4 Limitations

One of the limitations of the present study was a relatively low number of subject (n = 9). The present study also investigated only male subjects, so great care must be taken when extrapolating the result on females. Another limitation was that the acute N-M response was investigated only after a specific running HIT protocol (3 x 10 x 30) therefore; the results cannot be generalized to other activities, such as cycling or xc skiing and etc. In addition, the effects of other running HIT protocols on the N-M performance are beyond the scope of this thesis. Those protocol specific responses could be investigated by future studies.

8. PRACTICAL APPLICATIONS AND CONCLUSIONS

The present study demonstrated that HIT training can be successfully used for improving endurance running performance. In addition, HIT training can be combined with low frequency strength training in order to increase strength performance without significant gains in muscle mass which might be a desirable adaptation for endurance athletes.

The present study also demonstrated that endurance athletes may experience a significant acute potentiation of jumping performance following exhaustive HIT training. This highlights the importance of having high intensity exercises in warm up protocols preceding jumping drills or tests. In addition, the findings of the present study demonstrate that the CMJ performance after exhaustive endurance training can be used as an easy-to-perform test reflecting muscular fatigue tolerance and PAP in endurance running. For example, if an athlete experienced PAP in a CMJ after HIT exercises and some weeks later the same athlete did not experience PAP with no changes in his maximal aerobic speed and VO2max, this could be interpreted as an impairment with his muscular capabilities with no changes in his metabolic adaptations (Boullosa et al 2011).

The findings of the present study could be also used in a training program design as it was shown that endurance trained individuals experienced an acute decrease in maximal isometric force capacity of lower limbs following HIT session. However, 24 hours seemed to be enough to recover from neuromuscular fatigue following exhaustive HIT session.

In conclusion, the present study indicated that isometric MVC capacity and muscle AL of lower extremities can be significantly impaired after the HIT running session, while jumping ability can be enhanced due to PAP in endurance trained athletes. The magnitude of the changes is highly individual. However in 24 hours post session PAP was not observed any more as well as MVC and AL impairment, meaning that PAP has only an acute effect and that 24 hours seemed to be enough to recover MVC and muscle AL capacity. The levels of NM fatigue were independent of the aerobic capacity of the subjects and no significant correlation was found between NM performance and running

time trail performance. The present study also showed that HIT combined with low frequency strength training is an effective training strategy for improving both running performance and maximal lower body strength in recreational male endurance runners.

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