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ABSTRACT

The present series of studies were designed to investigate the regulation of leg stiffness during stretch-shortening cycle (SSC) exercise. More specifically, this thesis examined the neuromuscular mechanisms affecting stiffness regulation at joint and muscle level, both in non-fatiguing and fatiguing SSC conditions. The results suggest that ankle joint, particularly the gastrocnemius (Ga) muscle, plays a key role in leg stiffness modulation both in non-fatiguing and fatiguing SSC conditions. It was also observed that the interaction between muscle fascicles and tendinous structures during short-contact SSC muscle actions differ between the soleus (Sol) and Ga muscles, indicating muscle- and intensity-specific dependency. Centrally pre-programmed motor commands are suggested to play major roles in leg stiffness adjustments during SSC exercise with short ground contact time.

Exhaustive SSC exercise induced acute and long-term impairments in stiffness regulation that did not recover in one week in some cases. The results indicate that both central and peripheral fatigue mechanisms contribute to the decreased muscle performance and consequently, to stiffness modulation during the SSC exercise. The long-lasting impairments in muscle performance and stiffness regulation are likely related to exercise-induced muscle damage and the subsequent inflammation process. High muscle activation prior to and during the early phase of the ground contact seems to be a pre-requisite for efficient stiffness regulation that may also delay the exhaustion during intensive SSC exercise.

Key words: Stretch-shortening cycle, stiffness, muscle fatigue, muscle damage, pre-activation, stretch reflex

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ORIGINAL PAPERS

This thesis is based on the following papers, which will be referred to by their Roman numerals. In addition, some data not presented in these papers are also included.

I   Kuitunen S, Avela J, Kyröläinen H, Nicol C and Komi PV 2002. Acute and prolonged reduction in joint stiffness in humans after exhausting stretch-shortening cycle exercise. Eur J Appl Physiol 88: 107-116.

II   Nicol C, Kuitunen S, Kyröläinen H, Avela J and Komi PV 2003. Effects of long- and short-term fatiguing exercises on reflex EMG and force of the tendon-muscle complex. Eur J Appl Physiol 90:470-479.

III Kuitunen S, Avela J, Kyröläinen H and Komi PV 2004. Voluntary activation and mechanical performance of human triceps surae muscle after exhaustive stretch-shortening cycle jumping exercise. Eur J Appl Physiol 91: 538-544.

IV   Kuitunen S, Kyröläinen H, Avela J and Komi PV 2007. Leg stiffness modulation during exhaustive stretch-shortening cycle exercise. Scand J Med Sci Sports 17:67-75.

V   Kuitunen S, Ogiso K and Komi PV 2010. Leg and joint stiffness in human hopping. Scand J Med Sci Sports (Accepted for publication).

CONTENTS

ABSTRACT
ACKNOWLEDGEMENTS
ORIGINAL PAPERS
CONTENTS

1 INTRODUCTION

As early as 1885, French physiologist Jules Marey noted that when two vertical jumps are performed successively the second one is consistently higher than the first one (Marey and Demeny 1885). This potentiation effect due to counter-movement action was later called as "wind-up" movement (Asmussen and Sorensen 1971) until Norman and Komi (1979) named it as stretch-shortening cycle (SSC). SSC is referring to a natural type of muscle action occurring in everyday activities (walking, running, jumping), where the active muscle lengthens prior to shortening action thus enabling greater force/power output as compared to pure shortening action alone. Considerable amount of research have been carried out to explain the mechanisms of the performance potentiation in during SSC muscle actions, both in isolated muscle specimens (e.g. Cavagna et al. 1965, 1968; Cavagna and Citterio 1974; Edman et al. 1978; Edman 1997; Ettema et al. 1990; Ettema 1996; Herzog and Leonard 2000) and *in vivo* human experiments (e.g. Aura and Komi 1986a,b; Bosco et al. 1982a,b; Cavagna et al. 1968; Svantesson et al. 1994; Thys et al. 1975).

SSC muscle actions have also been used to study neuromuscular fatigue mechanisms. Fatigue induced by SSC exercise has been shown to be a complex phenomenon, consisting of a combination of neural, metabolic and structural changes in neuromuscular system (Komi 2000; Nicol et al. 2006). Progression of SSC induced fatigue as well as the subsequent recovery depends on various factors, including e.g. type, intensity and duration of the SSC exercise. Typically, SSC fatigue induces a bimodal recovery pattern, with a quick partial recovery followed by a secondary decline in performance, when exhaustive SSC fatigue protocol is used.

Houk (1979) has proposed a widely accepted concept of stiffness (change in length relative to change in force) being the regulated property of the neuromuscular system. Therefore, the neuromuscular mechanisms involved in normal and fatiguing SSC muscle actions are closely related to stiffness regulation of the musculo-skeletal system. This thesis work attempts to clarify the neuromuscular mechanisms of stiffness regulation in SSC muscle actions.

2 REVIEW OF LITERATURE

2.1 Neuromuscular basis of stretch-shortening cycle (SSC)

2.1.1 Performance potentiation and utilization of elastic energy in SSC

It has been suggested that performance potentiation in SSC is likely related to storage and utilization of elastic energy within the muscles (Asmussen and Bonde-Petersen 1974; Cavagna 1977). Tendons are likely to play a major role in this regard since they are known to be able to return more than 90% of the stored energy (Alexander and Bennet-Clark 1977; Bennet et al. 1986; Ker 1981). It has been shown, however, that elastic energy can also be stored within the myofilaments during the cross-bridge cycle (Huxley and Simmons 1971; Rack and Westbury 1974). Several factors are contributing to performance potentiation during SSC muscle actions, including muscle stretching velocity (Asmussen and Bonde-Petersen 1974; Bosco et al. 1981), coupling time between stretching and shortening (Bosco et al. 1981; Komi 1983; Thys et al. 1972), muscle properties (Dyhre-Poulsen et al. 1991; Viitasalo and Bosco 1982), age and gender (Bosco and Komi 1980; Komi and Bosco 1978).

Apart from the pure mechanical advantage of tendon elasticity due its efficient recoil properties, it also allows muscle fibers to operate close to their optimal force-length region. *In vivo* measurements during SSC locomotion, both in animal studies (Griffiths 1991; Hoffer et al. 1989; Roberts et al. 1997) and human experiments (for reviews, see Fukunaga et al. 2002; Ishikawa and Komi 2008; Kawakami and Fukunaga 2006) have indicated that muscle fibers may actually work almost isometrically or even shorten during the ground contact phase, despite the lengthening of whole muscle-tendon complex (MTC). However, this fascicle-tendinous tissue (tendon and aponeurosis) interaction may differ between the movement tasks (Fukunaga et al. 2002; Ishikawa et al. 2007) and intensity (Ishikawa and Komi 2004) as well as between the muscles (Ishikawa et al. 2005a,b; Sousa et al. 2007). Recent observations indicate that fascicle-tendon behavior may also be affected by prolonged SSC exercise (Cronin et al. 2009) and aging (Hoffren et al. 2007; Mian et al. 2007).

2.1.2   Role of pre-activation in SSC

In the experiment of Melvill-Jones and Watt (1971), the authors observed that the gastrocnemius muscle was activated before the contact to the ground in landing and hopping movements. This was suggested to be a pre-programmed activation pattern, set by higher centers of nervous system, in order to compensate for the sudden change in external load due to contact to the ground (Melvill-Jones and Watt 1971). It is of particular importance in fast SSC movements with high external loading demands, such as running and rebound jumps (Asmussen and Bonde-Petersen 1974; Dietz et al. 1979; Grillner 1972; Moritani et al. 1991). Its activity is affected by the required output of the SSC task (Dyhre-Poulsen et al. 1991; Gollhofer and Kyröläinen 1991; Moritani et al. 1991). It has been suggested that high and well-timed pre-activation is a prerequisite for efficient SSC performance (Horita et al. 2002; Kyröläinen et al. 2003; Moritani et al. 1991). Kyröläinen and Komi (1995) also observed a faster rate of electromyography (EMG) development in power-trained athletes, as compared to endurance athletes, The reported faster rate of EMG development and consequent higher power output in power-trained athletes during drop jump, as compared to endurance athletes (Kyröläinen and Komi 1995), indicates possible adaptation of central nervous system (CNS) to training.

2.1.3   Role of stretch reflexes in SSC

In the experiment of Melvill-Jones and Watt (1971), the authors observed also a burst of EMG activity in Gastrocnemius (Ga) muscle around 50ms after sudden ankle dorsiflexion movements, including stepping and hopping. This monosynaptic reflex response was followed by a second activity burst at 120ms from the initiation of the stretch. The monosynaptic reflex response had virtually no effect on muscle tension, whereas the latter one, named as 'functional stretch reflex', was followed by a rise in force (Melvill-Jones and Watt 1971). The authors concluded that, taking into account the electro-mechanical delay (EMD) of about 50ms (Norman and Komi 1979), the functional stretch reflex would contribute to muscle force only at the late ground contact phase and the preceding activity would, therefore, be preprogrammed by CNS. In line with this, Grillner (1972) suggested that stretch reflex induced load compensation would occur too late during fast SSC locomotion activities. Later on, however, Dietz et al. (1979) were able to demonstrate an abrupt increase in Ga muscle activity, exceeding the EMG obtained during a maximal voluntary contraction, 35-45 ms after the beginning of ground contact in running. The authors suggested that the monosynaptic (spinal) stretch reflex may indeed have a significant contribution to muscle activation already during the early phase of ground contact and, subsequently, to the mechanical output even in fastest SSC locomotion (running). Considering the reported EMD values as short as 10-12 ms, obtained with direct tendon force recordings (Nicol and Komi 1999), the monosynaptic stretch reflex could contribute to mechanical output already during the stretching phase of SSC

muscle action. Although several studies support the functional significance of stretch reflex during SSC muscle actions (for further details, see Komi and Gollhofer 1997), its relevance in bouncing type locomotion has also been questioned (Hof 2003).

2.2 Stiffness regulation during SSC

Houk (1979) proposed that the regulated property of skeletomotor reflexes is muscle stiffness (Δforce/Δlength), instead of separately controlling muscle length and force. This 'motor servo' control system is based on the idea that the feedback combined from muscle spindles (excitation) and tendon organs (inhibition) tends to maintain the stiffness of muscle (e.g. Houk 1979). This idea is supported by experimental evidence from several studies with animals (Hoffer and Andreassen 1981; Nichols and Houk 1976) and humans as well (Allum and Mauritz 1984; Crago et al. 1976; Sinkjær et al. 1988). Stiffness regulation has been suggested to provide a spring-like interface between the body and external environment to counteract abrupt changes occurring during daily activities and natural locomotion (Houk 1979). This chapter focuses on the stiffness of musculoskeletal system at different levels with a special emphasis on SSC muscle actions.

2.2.1 Muscle and tendon stiffness

Stiffness of MTC is determined by elastic properties of contractile and elastic (serial and parallel) components. *In vivo* human muscles, tendons are aligned in series with sheet-like tendinous structures surrounding bundles of muscle fibers (fascicles) called aponeurosis. Jewell and Wilkie (1958) observed that a significant part of the length change of tendinous structures occurs indeed in aponeuroses. The current knowledge indicates that in active muscles the aponeurosis is likely transferring the force to outer tendon, which possess higher capacity to storage and release of elastic energy (Magnusson et al. 2008). The tensile tests of isolated tendons indicate that tendon stiffness is relatively constant after the initial 'toe' region (see e.g. Butler et al. 1978). It is not clear, however, whether the mechanical properties differ between tendon and aponeurosis. Some authors have reported similar stiffness of free tendon and aponeurosis in animal experiments (Scott and Loeb 1995; Trestik and Lieber 1993). *In vivo* human experiments have revealed contradictory findings showing equal (Muramatsu et al. 2001), greater (Magnusson et al. 2003) or smaller strain of free tendon than in aponeurosis (Maganaris and Paul 2000b,c). Majority of data suggest greater stiffness in aponeurosis as compared to free tendon (Magnusson et al. 2008), although opposing findings have also been presented (Arampatzis et al. 2005; Maganaris and Paul 2000b,c). Furthermore, the current literature also reveals large inter-individual differences between the mechanical

properties of tendon and aponeurosis (Magnusson et al. 2008). Another open question is, whether the strain is uniform throughout the tendinous structures. Muramatsu and co-authors have reported similar strain between superficial and deep aponeuroses (Muramatsu et al. 2002a) as well as between proximal and distal parts of superficial aponeurosis (Muramatsu et al. 2002b). On the other hand, differential strain has also been observed between distal and proximal aponeurosis (Magnusson et al. 2001). Finni et al. (2003) have also reported non-uniform strain patterns in human soleus aponeurosis-tendon complex.

Tendinous structures are viscoelastic and, therefore, capable of storing elastic energy. Storage and recoil of elastic energy has been shown to play an important role in total external work during SSC activities (Kurokawa et al. 2003; Lichtwark and Wilson 2005; Voigt et al. 1995a,b). Experiments with various isolated mammalian tendons have revealed small hysteresis (3-20 %) indicating low energy dissipation during cyclic loading (Bennet et al. 1986; Ker 1981; Pollock and Shadwick 1994). The hysteresis values obtained in human tendons *in vivo* show similar values (mean of 17-26%, range 5-36%) (Kubo et al. 2001; Kubo et al. 2005; Lichtwark and Wilson 2005; Maganaris and Paul 1999, 2000a, 2002; Zhao et al. 2009).

2.2.1.1 Short-range stiffness

Hill (1968) observed that a resting muscle has some 'active' tension operating at very small range due to some 'short-range elastic component' lying between the myofilaments. Similar effect was found in active muscle, where forcible stretching of the muscle caused an initial steep rise in force with subsequent slow rise or even force reduction (Joyce et al. 1969). This resistance due to active cross-bridges was named as 'short-range stiffness' by Rack and Westbury (1974). Short-range stiffness covers only about 3-4 % of the change in muscle length (Rack and Westbury 1974) after which muscle yields due to detachment of active crossbridges (Flitney and Hirst 1978). In human muscles *in vivo*, it has been shown that the short-range stiffness is of contractile origin and that muscle (contractile) stiffness is greater than tendon stiffness in response to ankle joint perturbartions up to 7 degrees during quiet standing (Loram et al. 2007). Regarding SSC tasks, Dyhre-Poulsen et al. (1991) reported that a high initial muscle stiffness at the beginning of the ground contact during landing and hopping movements is likely due to short-range stiffness. Furthermore, Gollhofer et al. (1992) suggested that lengthening of the muscle-tendon complex (MTC) during the eccentric phase of a drop jump likely exceeds the limits of short-range stiffness. On the other hand, recent studies recording the muscle fascicle length changes during SSC movements have reported isometric or even shortening behavior of muscle fibers during the eccentric phase of SSC action (for reviews, see Ishikawa and Komi 2008; Kawakami and Fukunaga 2006). This indicates that muscle fibers may actually operate within the short-range stiffness region even during high force SSC tasks.

2.2.2 Joint stiffness

Stiffness of a single joint is determined by all the muscles, ligaments and other structures crossing the joint. Passive joint stiffness (when all the muscles crossing the joint are relaxed) is fairly small, about 19 Nm/rad in ankle joint (Gottlieb and Agarval 1978; Kearney and Hunter 1982), and does not play a significant role in natural SSC movements, where ankle joint stiffness reaches values ranging from 400 to 4500 Nm/rad (Arampatzis et al. 2001; Farley et al. 1998; Farley and Morgenroth 1999; Hobara et al. 2009; Kuitunen et al. 2002). During the early phase of joint perturbation the joint stiffness is determined mainly by intrinsic mechanical properties of muscle (intrinsic stiffness) (e.g. Blanpied and Smidt 1992). Several studies have shown that intrinsic stiffness increases with increasing joint torque/muscle activation (Sinkjær et al. 1988; Weiss et al. 1988) and is relatively independent of the perturbation amplitude, apart from the initial high stiffness phase (short-range stiffness) discussed in the preceding chapter. After about 50 ms after the onset of joint perturbation (muscle stretch) the stretch reflexes start to contribute to stiffness due to reflex-induced increment in muscle activation (Allum and Mauritz 1984; Dietz et al. 1979; Melvill-Jones and Watt 1971). The contribution of 'reflex stiffness' to the total joint/muscle stiffness is reported to be of significant magnitude (Allum and Mauritz 1984; Hoffer and Andreassen 1981; Sinkjær et al. 1988). During natural SSC muscle action, Horita (2000) observed close coupling between between stretch reflex and stiffness regulation. Fatiguing SSC exercise induced a decrease in SSC performance that was related to decreased knee joint stiffness and accompanied by reduced stretch reflex activity of the vastus lateralis muscle.

2.2.3 Leg stiffness

In several natural SSC type activities, such as running and jumping, the center of mass of the body represents a spring-like behavior similar to a bouncing ball due to the elastic properties of muscles, tendons and ligaments. Therefore, a simple spring-mass model, consisting of a mass and a linear spring, has been introduced to study the stiffness regulation of leg/whole body in SSC activities (Blickhan 1989). Leg stiffness is usually calculated by dividing the peak ground reaction force (GRF) by compression of the leg spring. Some authors have also determined leg stiffness on the basis of period of oscillation of vertical GRF (Cavagna et al. 1988) or natural frequency of oscillation using contact and flight times (McMahon et al. 1987). McMahon and Cheng (1990) differentiated leg stiffness and vertical stiffness in running by different methods to calculate leg spring compression, the latter being defined by vertical displacement of the center of mass (CoM) and the first one using the actual change in leg length. In case of the vertical motion only (like hopping in place) leg stiffness obviously equals vertical stiffness.

2.3 SSC fatigue and stiffness regulation

Regardless of type and/or intensity of SSC muscle action it has been shown to result in decreased force/power output, when repeated long enough (for review see Nicol et al. 2006). SSC fatigue is a complex phenomenon consisting of various neural, mechanical and metabolic changes that depends on the SSC task itself. The SSC exercise-induced performance impairment often follows a bimodal recovery pattern with a quick recovery within 1-2 hours after the exercise and a subsequent secondary decline usually peaking at two days post exercise (Avela et al. 1999; Dousset et al. 2007; Faulkner et al. 1993; MacIntyre et al. 1996). Complete recovery after exhaustive SSC exercise may take more than a week (Nicol et al. 2006).

Although number of studies have shown reduced muscle performance after SSC exercise, the effects on stiffness regulation may vary. Using various SSC modalities, researchers have reported increased (Moritani et al. 1990; Rodacki et al. 2001), decreased (Avela and Komi 1998; Horita et al. 1996, 1999; Toumi et al. 2006) or unaltered (Hunter and Smith 2007; Padua et al. 2006; Slawinski et al. 2008) stiffness (of muscle, joint or leg) after SSC exercise. Possible underlying mechanisms for SSC exercise induced performance impairments have been discussed as follows.

2.3.1 Acute effects of SSC fatigue

Fatigue may occur at various sites of the neuromuscular system from excitatory input to higher motor centres to neuromuscular transmission (central fatigue) and further through sarcolemmal excitation to cross-bridge cycle action between actin and myosin (peripheral fatigue) (Bigland-Ritchie 1984). A recent review (Nicol et al. 2006), summarizing the outcome of 26 SSC fatigue studies, indicates a significant contribution of peripheral fatigue (mainly related to metabolic fatigue and exercise-induced muscle damage) to the observed performance reduction after SSC exercise.

2.3.1.1 Peripheral fatigue

Decreased muscle force/power output is commonly observed after repeated SSC muscle actions. This has been shown as impairments in SSC performance as well as in voluntary and evoked isometric strength tests (Nicol et al. 2006). Intensive SSC exercise may lead to a depletion of energy substrates as well as accumulation of metabolic by-products that are known to contribute to the decreased force output (for reviews, see Fitts 1994; Green et al. 1997). This type of peripheral fatigue, that has been shown to recover quickly after cessation of exercise and normalization of metabolites, is termed metabolic fatigue (e.g. Green 1997). Metabolic fatigue has been suggested to play a significant role in post-exercise performance impairments after intensive short-term SSC exercise

(Gollhofer et al. 1987a,b; Horita et al. 1996; Lattier et al. 2004; Nicol et al. 1996; Skurvydas et al. 2000). Accumulation of metabolic by-products of intense muscle activity (e.g. lactate) may contribute to the post-exercise force decrement directly by impairing the excitation-contraction coupling process (Chin et al. 1997) or indirectly through reduced stretch reflex sensitivity (Avela et al. 1999; Nicol et al. 1996). Horita (2000) suggested that the observed deterioration in knee joint stiffness regulation during SSC could have resulted partly from fatigue-induced impairments in stretch reflex function.

On the other hand, reduced force/power output has been observed after SSC exercise without a significant involvement of metabolic fatigue (Skurvydas et al. 2000). This has been clearly evidenced after eccentric exercise protocols (for a review, see e.g. Allen 2001) and may, therefore, account for part of the post-exercise force decrement after SSC exercise as well. Reduced Ca^{++} release by sarcoplastic reticulum (e.g. Favero 1999) and changes and/or damage of intracellular structures (Friden et al. 1981; Newham et al. 1983) have been proposed as possible mechanisms.

2.3.1.2 Central fatigue

Central fatigue refers to a reduction in voluntary activation of muscle during exercise (e.g. Gandevia 2001). The observed reduction in muscle force output after SSC exercise is often associated with reduced muscle activation (Nicol et al. 2006) indicating that central fatigue mechanisms may contribute to the post-exercise impairment in force/power output. This is particularly common after a long-term SSC exercise (running) (Avela et al. 1999; Komi et al. 1986; Millet et al. 2002, 2003; Nicol et al. 1991; Place et al. 2004; Racinais et al. 2007). In fact, Petersen et al. (2007) found decreased MVC force, but no impairment in evoked muscle force, after a marathon race in highly trained runners suggesting that central rather than periferal fatigue mechanisms were dominating after marathon. Similarly, central fatigue has been suggested to contribute to the performance impairment during intermittent intensive activities of 30 minutes or longer of duration (racket sports) (for a review, see Girard and Millet 2008). However, a short-term (< 10 mins) intensive SSC exercise is not likely to induce significant central fatigue (Lattier et al. 2004; Strojnik and Komi 1998, 2000). In fact, muscle activation has been reported to increase after maximal SSC exercise of short duration (Strojnik and Komi 1998). These findings fit well with the observations of Bigland-Ritchie and Woods (1984), who suggested that central fatigue may not limit muscle force production in intermittent submaximal contractions lasting up to 20 mins, although it may contribute to force decline already after 1 min of continuous (maximal) contraction.

Asmussen and Mazin (1978) originally suggested that the decline in voluntary activation could originate from the muscle itself through some reflex responses. This reflex inhibition was further supported by Bigland-Ritchie et al. (1986) and confirmed by Garland (1991) through activity of the group III and IV muscle afferents. These small muscle afferents are known to be sensitive to several parameters related to e.g. metabolic fatigue (Dercherchi and Dousset

2003) and/or muscle damage (Kniffki et al. 1978; Rotto and Kaufmann 1988) commonly observed after SSC exercise. Duchateau and Hainaut (1993) suggested that metabolic stimulation of these small muscle afferents may lead to presynaptic inhibition of the Ia terminals and/or inhibition of interneurones. Another proposed hypothesis relies on reduced Ia-activity (disfacilitation) of α-motoneuron pool due to withdrawal of fusimotor support to muscle spindles (Bongiovanni and Hagbarth 1990; Macefield et al. 1991), fatigue of intrafusal fibres (Emonet-Dénand and Laporte 1974; Fischer and Schäfer 2005) and/or mechanical unloading of muscle spindles (Fowles et al. 2000; Avela et al. 2004). The possible role of supraspinal fatigue in reduced neural drive (Brasil-Neto et al. 1993, 1994) has also been suggested after various sports activities (Höllge et al. 1997; Ross et al. 2007; Verin et al. 2004), although its contribution in dynamic muscle contractions has been questionned (Löscher and Nordlund 2002).

2.3.1.3 Mechanical changes in muscle-tendon complex (MTC)

Increased muscle stiffness is a common observation after eccentric exercise (Howell et al. 1993; Jones et al. 1987; Whitehead et al. 2001). It has been proposed that injury contractures occur in damaged muscle fibers after eccentric exercise loading thus increasing the passive muscle tension (Whitehead et al. 2003). Previous studies using SSC fatigue protocols have reported both an increase (Ishikawa et al. 2006) and a decrease in plantarflexor torque during passive dorsiflexion movement after exhaustive SSC exercise (Avela et al. 1999) indicating changes in MTC stiffness after SSC exercise as well. In addition to the proposed injury contracture theory as a cause of increased muscle stiffness (Whitehead et al. 2003), long-term SSC exercise may also modify the compliance of tendinous structures in order to reduce the muscle stiffness immediately after the exercise (Avela et al. 1999, 2004). Reduced stiffness of Sol muscle has also been reported during SSC muscle action after fatiguing SSC exercise (Avela and Komi 1998). The observed reduction in Sol stiffness was found to be significantly correlated to decreased pre-activation (Avela and Komi 1998).

2.3.2   Delayed effects of SSC fatigue

Performance impairments after SSC exercise last usually several days (e.g Nicol et al. 2006). This indicates that other mechanisms than metabolic fatigue are contributing to the observed long-lasting impairments. SSC exercise has been shown to induce contractile failure of muscle (indicated by electrically evoked contractions), which exhibits a slow gradual recovery over several days (Skurvydas et al. 2000, 2006). However, recovery of voluntary and reflex force as well as their EMG manifestations after exhaustive SSC exercise often shows a bimodal recovery pattern with an initial post-exercise decline followed by a quick partial/complete recovery within 1-2 hours and a subsequent secondary decline 2-4 days after the exercise (Avela et al. 1999; Dousset et al. 2007; Nicol et al. 1996). This bimodal recovery pattern is typically observed after pure

eccentric exercise protocols both in animals (Faulkner et al. 1993) and humans (MacIntyre et al. 1996; Paulsen et al. 2010).

The rapid recovery of neuromuscular parameters after SSC exercise is thought to be due to recovery of metabolic fatigue (e.g. Nicol et al. 2006). The secondary decline in force and EMG parameters has been suggested to result from exercise-induced muscle injury and subsequent inflammation process (e.g. Armstrong et al. 1991). As early as 1902, Hough observed a delayed muscle pain after concentric-eccentric contractions of finger muscles and suggested that some sort of rupture occurs within the muscle during the exercise (Hough, 1902). This has been later verified by morphological changes in muscle biopsies after eccentric exercise in humans (Friden et al. 1981; Newham et al. 1983) indicating that the initial event of muscle damage may be mechanical in nature. This initial damage leads to increased level of intracellular Ca^{++} (Balnave and Allen 1995; Ingalls et al. 1998; Lynch et al. 1997; Westerblad et al. 1993) and further to degradation of intracellular proteins and invasion of phagocytic and inflammatory cells into the site of muscle injury within few hours after the exercise (e.g. Armstrong et al. 1991). Some of the metabolic substances related to muscle damage and subsequent inflammation are known to stimulate the discharge of group III and IV small muscle afferents (Kniffki et al. 1978; Mense and Meyer 1988; Rotto and Kaufmann 1988). The metabolic sensitization of these small muscle afferents could lead to presynaptic inhibition and further to a reduction in neural drive and muscle force (Duchateau and Hainaut 1993). This mechanism has been suggested as a potential candidate for the observed delayed secondary reduction in neural parameters after fatiguing SSC exercise (Dousset et al. 2007; Nicol et al. 1996; Avela et al. 1999). In addition to this metabolic hypothesis, the secondary decline in performance may partly result from mechanical and/or architectural changes in MTC itself (namely due to increased muscle volume caused by swelling and possible changes in tendinous tissue stiffness), as reported by Ishikawa et al. (2006). Nevertheless, the current literature related to SSC fatigue reveals the complexity of SSC fatigue mechanisms that are not completely understood yet.

3   PURPOSE OF THE STUDY

Lower extremity stiffness plays an important role in musculoskeletal performance in SSC locomotion (Butler et al. 2003). However, the current literature is still somewhat controversial regarding the neuromuscular mechanisms of leg stiffness adjustments in natural SSC muscle actions. More specifically, how do we adjust the stiffness of the individual "springs" (muscles, tendons, joints) of the leg system in order to meet the required movement mechanics? The present thesis work was focused to clarify the central and peripheral mechanisms related to stiffness regulation both in normal (unfatigued) and fatiguing SSC conditions. Since the earlier studies have indicated that ankle joint stiffness plays an important role in leg stiffness adjustment (Farley et al. 1998; Farley and Morgenroth 1999; Horita 2000; Arampatzis et al. 2001) a special emphasis was put on the function of plantarflexor muscles in leg/joint stiffness adjustments. The detailed aims of the present work are outlined as follows:

1)   In a system with multiple springs the least stiff spring will undergo greatest displacement in response to a certain force, thus having the greatest influence on the overall stiffness of the system. Some of the earlier studies regarding human SSC type locomotion indicated that the ankle is the least stiff joint in the leg system (Arampatzis et al. 2001; Günther and Blickhan 2002; Kuitunen et al. 2002), whereas some other studies have found higher stiffness in the ankle than in the knee joint (Farley et al. 1998; Arampatzis et al. 1999; Farley and Morgenroth 1999; Rapoport et al. 2003). The earlier PhD thesis of Horita (2000) indicated that ankle joint kinematics plays also an important role in leg stiffness regulation during fatiguing SSC exercise. The present study was focused to clarify the stiffness adjustment of the ankle and knee joints in SSC exercise both with (I, III) and without fatigue (V). It was hypothesized that leg stiffness is more closely related to ankle than knee joint stiffness in SSC exercise both with and without fatigue. (I, III, V)

2) Pre-landing muscle activation and stretch reflexes has been suggested to contribute to joint and leg stiffness regulation during SSC locomotion (Gollhofer et al. 1984, Horita et al. 1996 1999). However, Farley et al. (1998) suggested that ankle joint stiffness in hopping is not adjusted by changing the level of muscle activation. Furthermore, the contribution of stretch reflexes to muscle activation and force in fast-contact SSC locomotion has been questioned (Hof 2003). Therefore, our focus was to examine the role of pre-activation and stretch reflexes in joint stiffness regulation during fast-contact SSC locomotion. (V)

3) In maximal short-contact bouncing type SSC actions muscle fascicles may even shorten during the ground contact phase, despite the lengthening of the MTC (Ishikawa et al. 2003, 2005b), thus indicating high muscle fibre stiffness in this type of SSC locomotion. Furthermore, it has also been suggested that this muscle fascicle behaviour may be muscle-dependent referring to possible differences between the one- and two-joint muscles (Ishikawa et al. 2005b). The purpose of the study was to examine the possible muscle- and intensity-dependent differences in the muscle fibre behaviour of the triceps surae muscle in short-contact bouncing-type SSC task with a special reference to stiffness regulation. It was hypothesized that the fascicle behaviour of Ga muscle is more affected by changes in performance intensity than Sol.

4) It is known that SSC exercise may induce both acute and long-term impairments in SSC performance (for a review see Nicol and Komi 2003). The acute impairments in SSC performance has been suggested to be associated with metabolic fatigue (for a review see Green 1997), when high-intensity SSC exercise protocol is used. On the other hand, impaired SSC performance has been also observed after low-intensity long-lasting SSC exercise without any significant metabolic fatigue (Avela et al. 1999, Nicol et al. 1991). Reduced neural drive with concomitant decrease in stretch reflex sensitivity has been suggested to play a role in this regard (Avela et al. 1999, Nicol et al. 1996). The long-term impairments in SSC performance has been suggested to be in association with exercise-induced muscle damage and the subsequent inflammation processes (e.g. Nicol and Komi 2003). The present objective was to further clarify the role and time course of these central and peripheral adaptation mechanisms in exhaustive SSC exercise and their effects on muscle performance and stiffness adjustments in SSC muscle actions. It was hypothesized that, in addition to peripheral fatigue, central fatigue may have a significant contribution to the post-exercise impairment in muscle performance and stiffness regulation also after high-intensity SSC exercise performed until complete exhaustion. (I, II, III, IV)

4 RESEARCH METHODS

4.1 Subjects

A total of 32 subjects participated in these studies. Table 1 presents the physical characteristics of the subjects in each experiment. The subjects were physically active young people involved in various sports. They were informed of the risks and benefits associated with the experiments and gave their written consent. The procedures used in these studies were accepted by the Ethical Committee of the University of Jyväskylä and followed the recommendations of the Declaration of Helsinki.

TABLE 1   Physical characteristics (Mean±SD) of the subject groups.

	Experiment 1 (n=9)	Experiment 2 (n=6)	Experiment 3 (n=8)	Experiment 4 (n=9)
Age (yrs)	25±3	26±7	29±5	28±4
Height (m)	1.80±0.08	1.83±0.09	1.80±0.07	1.78±0.04
Weight (kg)	78±10	75±11	80±8	76±6
Original paper	I-IV	II	V	

4.2 Experimental design

Four separate experiments were carried out in this series of studies. The first experiment focused on the central and peripheral mechanisms related to stiffness regulation in fatiguing SSC conditions on a special sledge apparatus (Fig. 1) (I-IV). In the second experiment, which was a continuation of the first one, fatigue was induced by a 10-km run (II). The third experiment was designed to address the stiffness adjustment of the ankle and knee joints in

bouncing type SSC exercise (IV). In the fourth experiment, direct recording of muscle fascicle length changes was combined with the conventional force and EMG measurements in order to get closer insight into the stiffness regulation at the level of muscle fibres and bundles.

FIGURE 1   The sledge apparatus for jump test. The subjects' lower limbs were supported by a rope to minimize the metabolic fatigue of the quadriceps muscle group during the airborne phase due to the inclined leg position.

4.2.1 Experiment 1 (I-IV)

In the first experiment, we modified the SSC fatigue protocol originally introduced by Horita et al. (1996) and Nicol et al. (1996). Consequently the present experiment, the subjects were fatigued until complete exhaustion by performing rebound jumps on a special sledge ergometer (Kyröläinen and Komi 1995) inclined at 24.9 degrees from the horizontal. They were seated on the sledge, and after a familiarization phase they were dropped from different starting heights to determine the optimal dropping height that led to their highest rebound performance (for details see Kyröläinen and Komi 1995; Nicol et al. 1996). Differing from those previous studies the protocol started with one hundred single maximal drop jumps from the optimal dropping height that were repeated every five seconds. This was done to induce muscle damage by increasing heavy eccentric loading component to the protocol without significant metabolic loading. This series of jumps was immediately followed by a continuous rebounding exercise (Horita et al. 1996; Nicol et al. 1996) to a submaximal height representing 70% of their maximal initial rebound performance. Unlike in the original protocol, the submaximal jumping in the present study was continued beyond the endurance limit until complete exhaustion, when the subjects were not able to jump at all. The purpose of this was to examine, if decreased voluntary activation is likely to contribute to

reduced performance also during intensive short-term SSC exercise as has been earlier observed in long-term SSC exercises (Avela et al. 1999; Millet et al. 2003; Place et al. 2004). In the present SSC exercise, the lower limbs were supported by a rope attached to the ceiling on the one end and surrounding the subject's ankles with a cushioned belt on the other end (Fig. 1). This procedure was done to minimize the metabolic fatigue of the quadriceps muscle group during the airborne phase due to the inclined leg position. The supportive system did not restrict the normal range of movement during the jumps.

Prior to the SSC fatigue passive stretch reflexes (PSR), maximal Hoffmann reflex (H-reflex) and compound mass action potential (MaxM) responses of Soleus (Sol) muscle, as well as plantarflexor torque during maximal isometric voluntary contraction (MVC) and low frequency (LF) electrical stimulation (20 Hz) were measured from the left leg. For these measurements, the subjects were seated in an ergometer chair with their left thigh clamped and the foot mounted on the motor-driven platform (Fig. 2). The subjects performed also maximal drop jumps from 35 and 55 cm heights and submaximal hopping on a force plate before the SSC fatigue protocol. These measurements were repeated immediately after as well as 2 hours, 2 days and 7 days post exercise.

FIGURE 2   The ankle ergometer for assessing neuromuscular performance of the plantar flexor muscles.

In addition to the ankle ergometer data, ground reaction forces (GRF) on the force plate and on the sledge apparatus, sledge seat position as well as the EMGs of Sol, gastrocnemius medialis (GaM), tibialis anterior (TA), vastus lateralis (VL) and biceps femoris (BF) were collected simultaneously with the high-speed video recording. Inverse dynamics were used to calculate joint moments during the jumping trials. Joint moments and sledge force were further plotted against the joint angular displacement and sledge seat displacement in calculating the joint and leg stiffnesses.

4.2.2 Experiment 2 (II)

In the second experiment six subjects performed a 10-km run on a 200-m indoor track at individually determined constant speed, which averaged 3.2 (0.2) m·s^{-1}. The running speed was based on the subject's estimation of his maximal performance over 10-km distance. The speed was controlled by a pacemaker (Proton, Naakka, Finland) and monitored at each lap with photocells over a 20-m distance.

4.2.3 Experiment 3 (V)

The third experiment was designed to examine ankle and knee joint stiffness regulation in short-contact bouncing type SSC exercise. The subjects performed bilateral hopping on a force plate at various intensity levels that were determined by peak vertical GRF corresponding to 3, 4, 5 and 6 times their body weight (BW) (Fig. 3).
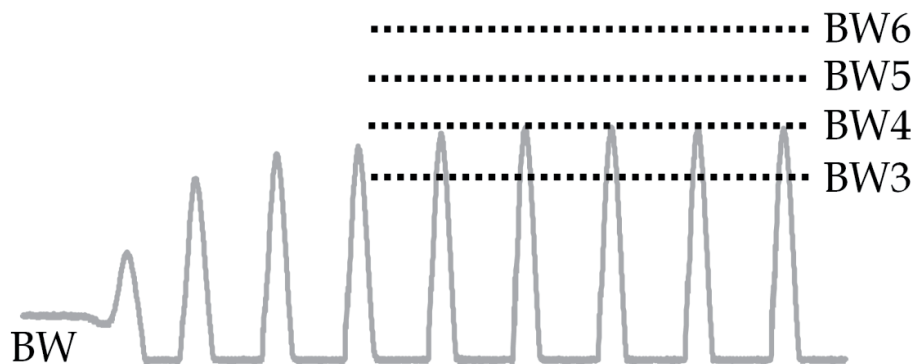
FIGURE 3   Determination of hopping intensities for hopping by peak vertical ground reaction force (GRF) (V). The GRF curve and the target GRF level was displayed on the oscilloscope screen for monitoring the intensity during the hopping trials.

In addition to the GRF, the measurements included hopping kinematics and EMG of selected leg muscles. Leg stiffness was calculated by dividing the peak GRF by the vertical displacement of the center of mass. Inverse dynamics was used to calculate joint moments. The joint stiffness was determined as a ratio between the peak joint moment and angular displacement during the braking phase (Kuitunen et al. 2002).

4.2.4 Experiment 4

In the fourth experiment, the hopping protocol was similar to the one in experiment 3. The selected intensity levels were Low (corresponding to three times the BW), medium (Med, five times the BW) and maximum hopping (Max). In addition to the kinetic, kinematic and EMG measurements described above, we utilized real-time ultrasonography for recording muscle fascicle length changes of the Sol and GaM muscles in vivo during the hopping (Fig. 4).
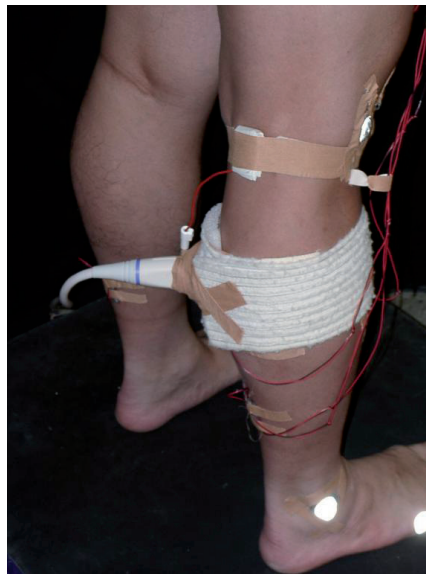
FIGURE 4   Ultrasound probe fixed onto the gastrocnemius medialis (GaM) muscle for recording of fascicle length changes in hopping.

4.3 Measurement procedures and analysis

4.3.1 Stiffness determination in SSC

4.3.1.1 Joint stiffness

For the joint stiffness determination in the drop jump and hopping tasks (I, V), GRFs were recorded synchronously with high-speed video (200 Hz). Reflective markers for video recording were placed on the distal head of the fifth metatarsal bone, heel, lateral malleolus, lateral epicondyle of the femur, greater trochanter and shoulder. From the video recordings, body segment coordinates were digitized (Motus software, Peak Performance Technologies, Inc., Denver, USA), filtered (Butterworth filter, cut-off frequency 20 Hz) and transferred to the computer system (SGI O2 R5000, Silicon Graphics, Inc., California, USA) for

further analysis. The scaled coordinates and GRF were synchronized and joint moments were calculated at 200 Hz (Belli et al. 2002). Anthropometric data, provided by the standards of Demster (1955), were used to determine inertia and mass of the segments. Four to ten successful trials were averaged in each jumping condition. The average joint stiffness (JS) for the ankle and knee was calculated as a change in the joint moment (ΔM) divided by the change in joint angle ($\Delta\theta$) in the braking phase (Fig. 5) (Kuitunen et al. 2002).

Joint stiffness (JS) calculation

$$JS = \frac{\Delta M_{BRAKING}}{\Delta\theta_{BRAKING}}$$
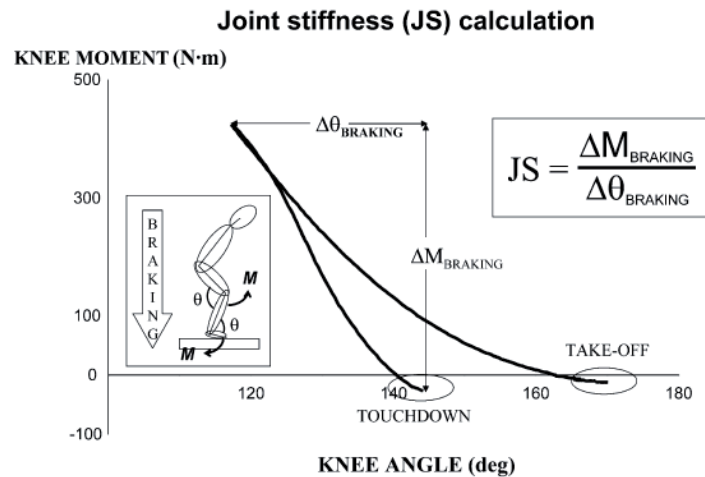
FIGURE 5   Knee joint moment – joint angle relationship during the ground contact phase in drop jump. Joint stiffness for both ankle and knee joints was calculated as an average value in the braking phase by dividing the change in joint moment ($\Delta M_{braking}$) by the respective change in joint angle ($\Delta\theta_{braking}$). The direction of positive joint moments and definition for joint angles are marked on the insert figure.

4.3.1.2 Leg stiffness

Leg stiffness was calculated similarly to earlier studies by dividing the peak GRF by the vertical downward displacement of the CoM (V) (calculated from the video data) and expressed as $kN \cdot m^{-1}$ (Ferris and Farley 1997; Farley and Morgenroth 1999; Hobara et al. 2008). For the leg stiffness determination in sledge jumps (IV), the contact phase was first divided into braking and push-off phases according to the sledge position signal. Leg stiffness was then calculated during the braking phase by dividing the reaction force at the end of the braking phase (F_{peak}) by the corresponding sledge seat displacement.

4.3.2 Muscle and tendon length and force measurements

4.3.2.1 MTC length determination

Joint angle data was used to estimate the MTC lengths of Sol and GaM according to the model of Hawkins and Hull (1990) by applying the individual segment lengths of the lower leg to the calculations.

4.3.2.2 Muscle fascicle and tendinous tissue (TT) length determination

Fascicle length changes of the Sol and GaM muscles were obtained by real-time ultrasonography (SSD-5500, Aloka, Japan). A 7.5 MHz linear array ultrasound probe of 6 cm long was fixed longitudinally in the midline onto the muscle belly of the GaM muscle. The probe was adjusted with care for obtaining a proper image of both the Sol and GaM fascicles. The probe was fixed with a foam plastic bracket and secured by tape and an elastic band wrapped around the lower leg. Ultrasonographic images of the muscle fascicles were transferred to the computer at 96 Hz. The ultrasonographic images, high-speed video and GRF data were synchronized with an electronic pulse signal.

The fascicle lengths of Sol and GaM were measured as the diagonal lengths between the deep and superficial aponeurosis (Fig. 6). The length of the tendinous tissue (TT) (outer tendon and the aponeuroses) were calculated as follows: TT length = MTC length – fascicle length * cos α (Fukunaga et al. 2001; Kurokawa et al. 2001, 2003; Ishikawa et al. 2003, 2005a,b), where cos α is the angle between the fascicle and deep aponeurosis. In case that the whole fascicle could not be visualized throughout the trial, the entire length of the fascicle was estimated trigonometrically (Finni et al. 2001). Fascicle and TT length changes were analyzed from two representative jumps in terms of the peak GRF and contact time. The reliability and reproducibility of the fascicle length measurement have been reported elsewhere (Finni et al. 2001 2003; Fukunaga et al. 1997 2001; Ishikawa et al. 2003; Kawakami et al. 1998; Kurokawa et al. 2003).
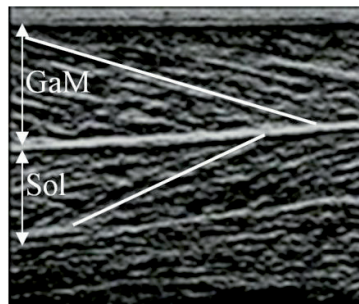
FIGURE 6   Ultrasonographic image of the gastrocnemius medialis (GaM) and soleus (Sol) muscles. The diagonal white lines show the fascicles of GaM and Sol in between the aponeuroses.

4.3.2.3 Estimation of Achilles tendon force (ATF)

Achilles tendon force (ATF) was calculated as the net joint moment divided by the instantaneous tendon moment arm that was estimated on the basis of Rugg et al. (1990). ATF was further divided into tendon forces of Sol and GaM in relation to their physiological cross-sectional areas (PCSA) corresponding to 56.2 and 15.4 %, respectively, of the total plantar flexor PCSA (Fukunaga et al. 1996).

4.3.3 Electromyography (EMG)

For recording the muscle EMG, bipolar surface electrodes (Beckman miniature skin electrodes 650437, Illinois, USA) with the fixed inter-electrode distance of 20 mm were placed on the selected muscles, including Sol, GaM and gastrocnemius lateralis (GaL), TA, VL and BF muscles of the right leg according to the recommendations of SENIAM (Hermens et al. 2000). For MVC and reflex assessments (see 4.3.4) electrodes were also placed on Sol and GaL of the left leg (I, II, III). The position of the electrodes was carefully marked on the skin with drawing ink for ensuring the same electrode location at the follow-up measurements (I, II, III, IV). The EMG electrode sites were shaved, abraded slightly with sandpaper and cleaned with alcohol for ensuring the low inter-electrode impedance (< 5 kΩ). The EMG signals were amplified and recorded telemetrically (ME3000P, MEGA, Kuopio, Finland; cut-off frequency 360 Hz/ 3 dB, CMRR >110 dB) to the computer simultaneously with the GRF data with a sampling frequency of 1000 Hz.

4.3.3.1 Stretch reflex component of EMG

The recorded EMG was first rectified and then averaged (aEMG) for the pre-activation, braking and push-off phases for evaluating the changes in muscle activity levels. For evaluating the contribution of the short-latency stretch reflex response (referred to M1 according to Lee and Tatton, 1978) to the EMG activity in the braking phase, aEMG was calculated as a 20 ms window starting at 30 ms for VL (Horita et al. 1996; Avela et al. 1999) and 45 ms for Sol, GaM and GaL (according to our pilot study; Ogiso 2003) after the beginning of ground contact (see also Fig. 9 in Results) (V). Correspondingly, aEMG for the background activity (BGA) prior to M1 response was calculated from 0-30 ms for VL and 0-45 ms for Sol, GaM and GaL (V).

4.3.3.1 EMG ratio

As has been suggested, high activation in the braking phase is advantageous for efficient storage of elastic energy (Asmussen and Bonde-Petersen 1974; Thys et al. 1975; Cavagna 1977; Aura and Komi 1986a), which can then be partly utilized in the subsequent push-off phase thus saving metabolic energy (Cavagna et al. 1964; Alexander and Bennet-Clark 1977; Alexander 1988).

Therefore, EMG ratio was calculated by dividing the integrated EMG (iEMG) of the braking phase by the iEMG of the push-off phase (Fig. 7) (Aura and Komi 1986a; Kyröläinen et al. 1998) This was used to evaluate the muscle activation patterns in terms of the efficiency of stiffness regulation (IV). This relative EMG ratio value allowed an inter-subject comparison in the EMG changes regardless of the magnitude of the absolute changes.
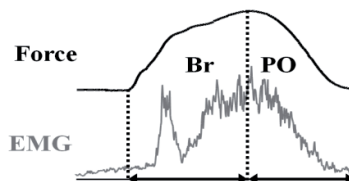
FIGURE 7   An example of reaction force (Force) and soleus EMG during a sledge jump. The contact phase was determined from the force and divided into the braking (Br) and push-off (PO) phases (vertical dotted lines) according to the sledge position signal. Integrated EMG (iEMG) was calculated for the corresponding phases and the EMG ratio was calculated by dividing the Br phase iEMG by the PO iEMG.

4.3.4 Assessment of neuromuscular properties of MTC

Voluntary and evoked plantarflexor torque as well as reflex measurements were performed with an ankle ergometer (Nicol et al. 1996; Avela et al. 1999). The subjects were seated in an ergometer chair with their left thigh clamped and the foot mounted on the motor-driven platform. Rotation axis of the ankle joint was aligned to correspond to that of the ankle ergometer platform. The initial ankle angle was 90 deg and the knee angle was set at 130 deg.

4.3.4.1 Maximal voluntary contraction (MVC) force and activation level (AL)

In the isometric MVC test, the MVC with the highest peak torque was chosen for the analysis and aEMG was calculated for a 500 ms window around the peak torque. In order to assess the level of the maximal voluntary activation (AL) during the MVC test, two consecutive supramaximal stimuli (pulse duration 1 ms, frequency 0.2 Hz, intensity of 25 % higher that was needed to induce MaxM) were superimposed during the plateau phase of plantarflexor torque (Fig. 8). The activation level was assessed by the twitch interpolation method (Merton 1954) according to the formula of Strojnik and Komi (2000): activation level $= 100 - D * (T_b/T_{max})/T_{tw} * 100$. An increase in MVC torque induced by the superimposed double twitch (T_b) was compared over the maximal voluntary torque (T_{max}) in proportion to the torque in maximal passive twitch response (T_{tw}). A correction factor D was used to take into account the possible difference between T_{max} and torque just before the double twitch stimuli.
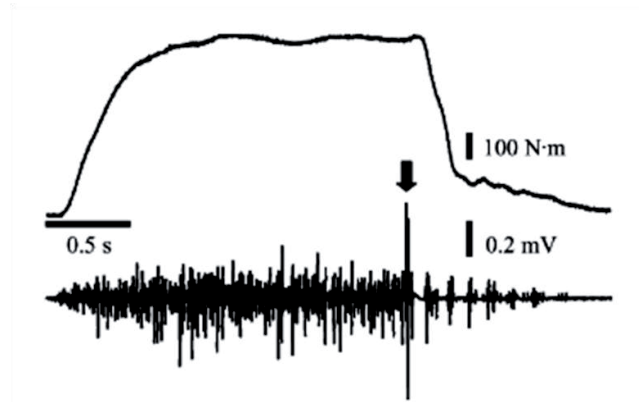
FIGURE 8   An example of maximal isometric voluntary contraction plantarflexion torque and EMG of the soleus (Sol) muscle. Supramaximal double twitch stimuli (arrow) were superimposed on the tibial nerve during the plateau phase of contraction for checking the level of voluntary activation.

4.3.4.2 Compound mass action potential (MaxM) and Hoffmann reflex (H-reflex)

For the electrical stimulation used in MVC (see 4.3.4.3), maximal Hoffmann reflex (H-reflex) and compound mass action potential (MaxM), stimulation electrode (pregelled Ag/AgCl electrodes, Niko, Denmark) was placed over the tibial nerve in the popliteal fossa and the anode superior to the patella. The position of the stimulating electrode was tested first in the standing position and then checked in the experimental position to ensure constant recording conditions. Single rectangular pulses of 1 ms duration were elicited (MEB-5304K, Nihon Kohden, Japan) when testing MaxM and H-reflex. Three highest peak-to-peak amplitudes of the Sol EMG response were calculated and averaged for the MaxM and H-reflex.

4.3.4.3 Electrically evoked force of MTC

For the low-frequency (LF) muscle stimulation (pulse duration 0.2 ms, frequency 20 Hz), two stimulating electrodes (5 cm x 5 cm; StimRode, Axelgaard Manufacturing Co., California, USA) were placed percutaneously on the proximal head of GaM and GaL muscles and one anode was located distally on the muscle-tendon region onto Sol. The stimulation intensity in LF test was set at three times the motor threshold. The LF torque was determined as an average torque during the plateau phase of LF stimulation. In all the evoked contractions, stimulation intensity was determined before every testing occasion.

4.3.4.4 Passive stretch reflexes

In PSR measurements of Sol muscle, ten consecutive dorsiflexions of 10 deg in amplitude with a stretching velocity of 110 deg s^{-1} were induced (I, III). In the experiment two, the PSR test consisted of two consecutive dorsiflexions of 2.8 deg in amplitude and 120 deg s^{-1} in velocity. Three different time intervals between the consecutive dorsiflexions were used. The longest plateau phase lasted 500 ms so that the mechanical (twitch) response was completed before the second dorsiflexion. To test MTC resistance to stretch during the dorsiflexion stimulus the other two stimuli were individually adjusted to start approximately at 30 and 70 % levels of the peak torque. The randomised series of stimuli included a total of 28 repetitions with 4-s intervals. The two latter ones were repated 7 times each, whereas the stimulus with longest plateau phase included 14 repetitions (II).

In the PSR test, peak-to-peak EMG amplitude of the Sol was determined and the responses were averaged for each subject. The first PSR response was excluded from the analysis to minimize the effect of muscle thixotropy (Proske et al. 1993) (I, III). From the mechanical twitch response, peak torque (PT) was calculated for each dorsiflexion condition (II). Mean torque (MT) was calculated as a difference between the PT and the torque at the end of 500-ms plateau phase (passive resistance to stretch, PSR). This method allowed a differentiation of the pure reflex-induced twitch response (Nicol et al. 1999). In the shorter plateau conditions, the second stretch did not cause any reflex EMG response and allowed calculation of the twitch resistance to stretch (TRS) by subtracting the PT during the longest plateau condition from the PT in double stretch during the short plateau conditions. Mean relative relaxation rate (RR) was determined as a torque decrement in 100 ms from the 90% level of the peak torque.

4.3.5 Blood analysis

Capillary blood samples from the fingertip were taken to determine blood lactate (B-La) level Pre and Post the SSC exercise. B-La was analysed enzymatically by using a commercial Lactate enzymatic kit (Biochemica Boehringer, Mannheim, Germany). Furthermore, venous blood samples were taken from the antecubital vein Pre, Post, 2h, 2d and 7d post exercise for determining serum creatine kinase (CK) activity. CK activity was analysed by using a CK enzymatic test kit (Biochemica Boehringer, Mannheim, Germany).

4.3.6 Statistical methods

Means and standard deviations (SD) or standard errors of means (SEM) were calculated for each subject group. Kolmogorov-Smirnov test (I, III, IV) and Shapiro-Wilk's tests (V) were used to examine the normality of data distribution and, if failed, non-parametric tests were used for statistical analysis. Friedman's non-parametric test for several related samples (I, III) and 1-

ANOVA for repeated measures (II, V) were used to examine, if there were differences between the measurements or conditions. If significant differences were found, non-parametric Wilcoxon's test (I, III, IV) and Tukey's post hoc test (V) were used to examine the significance of difference between two measurements or conditions. For experiment II, Student's *t*-test for independent samples was used for subgroup comparisons and paired *t*-test for B-La and CK. Non-parametric Spearman's correlation coefficients (I, III, IV) and Pearson correlation coefficients (V) were calculated for examining possible relationships between the selected parameters. The level of significance was set to P<.05 for all the tests.

5   RESULTS

This chapter summarizes the main findings of the present experiments. Original papers (I-V) should be consulted for more details. Some unpublished results are also included (Experiment 4).

5.1 Effects of exercise intensity on leg and joint stiffness regulation

5.1.1 Joint and muscle mechanics and stiffness modulation

Increasing the hopping intensity was accompanied by a decrease in hopping frequency and ground contact time as well as by an increase in flight time. Fig 9 shows the basic response curves of these jumps with related GRF, CoM and EMG patterns. Despite the twofold increase in peak GRF with increasing hopping intensity from BW3 to BW6 condition, leg stiffness remained essentially the same (Fig. 10). Vertical displacement of CoM behaved in a similar manner showing greater displacement with increasing hopping intensity. Parallel to the changes in GRF and CoM displacement, ankle joint moment showed also a twofold increase between BW3 and BW6. This was accompanied by an increase in angular displacement with increasing hopping intensity from BW3 to BW6 (Fig. 11, left panels). No changes were, therefore, observed in ankle joint stiffness either (Fig. 10, lower panel).

Different from the responses of the leg and ankle joint stiffness, knee joint stiffness increased significantly with increasing hopping intensity (Fig. 10, lower panel). For the knee joint, a five-fold increase was found in the joint moment that was accompanied by twofold increase in joint angular displacement with increasing hopping intensity from BW3 to BW6 (Fig. 11, right panels). Furthermore, knee angle at touchdown decreased significantly with increasing hopping intensity.
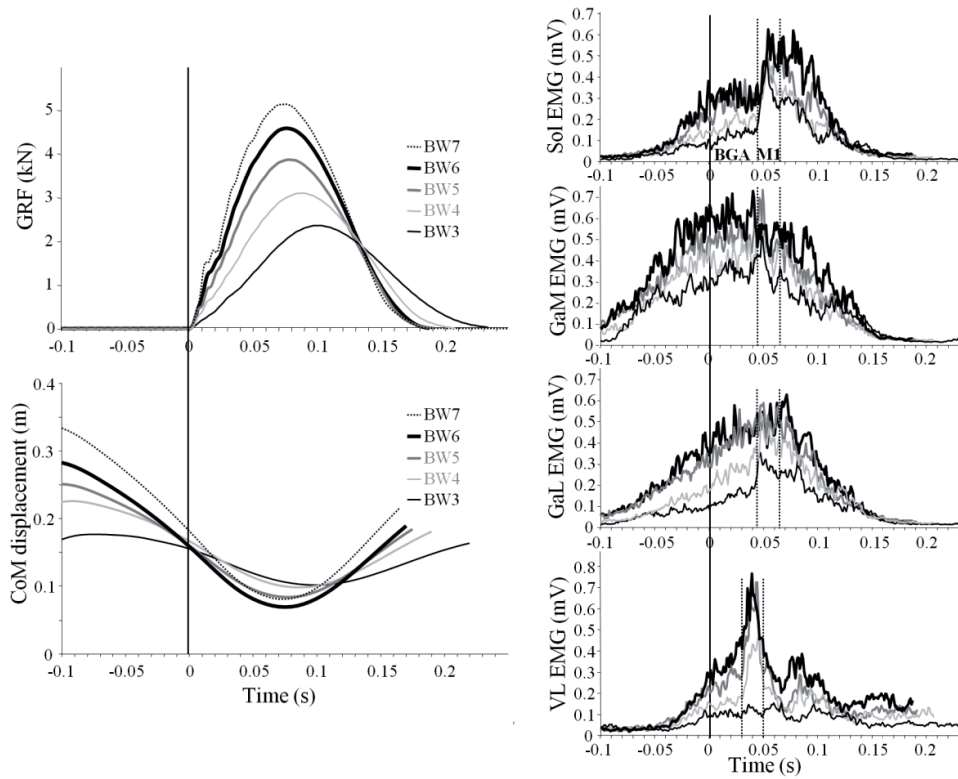
FIGURE 9   Vertical ground reaction force (GRF) and center of mass (CoM) displacement (left panels) together with rectified EMGs of the soleus (Sol), medial gastrocnemius (GaM), lateral gastrocnemius (GaL) and vastus lateralis (VL) muscles (right panels) plotted against time at the different intensity levels (BW3-BW7) in hopping. Data represent means of the subjects. Vertical solid line denotes the beginning of the ground contact. The dotted vertical lines represent the window for stretch reflex EMG calculation (M1). BGA, background activation
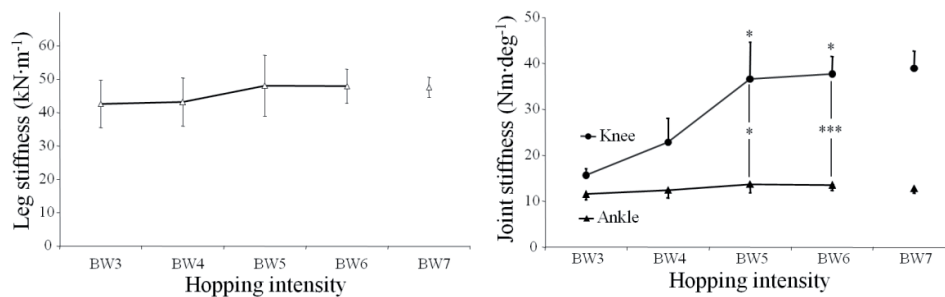
FIGURE 10   Leg and joint stiffness (Means±SD) of the knee and ankle at different hopping intensities. *,*** significantly different between the intensities / joints (P<.05, P<.001)
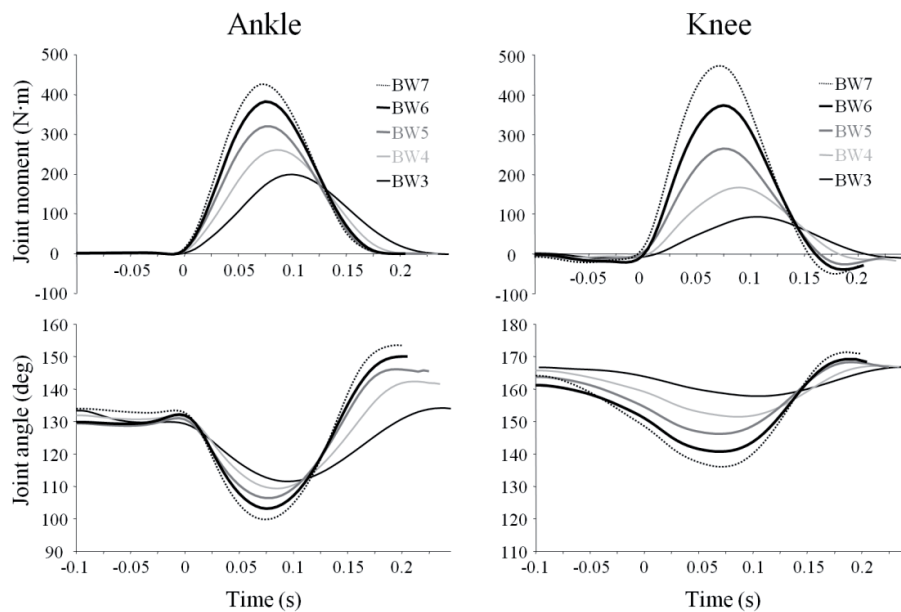
FIGURE 11   Joint moments and angles of the ankle and knee at different hopping intensities. Data represent means of the subjects.

5.1.1.1 Muscle fascicle – tendon interaction

Ultrasonographic imaging of muscle fascicle and tendinous tissue length changes of triceps surae MTC in hopping revealed a non-uniform fascicle behavior between Sol and GaM muscles and also between the hopping intensities. Sol muscle fascicles presented a stretch-shortening behavior after the initial shortening at the beginning of the ground contact at all hopping intensities. For GaM, the initial shortening of fascicles was greater and the subsequent lengthening occurred later than in Sol (Fig. 12, upper right panels). Furthermore, the end of the fascicle lengthening occurred close to the end of MTC lengthening for Sol, whereas in GaM the fascicle lengthening at Med and Max intensities continued after the MTC started to shorten already. Increasing the hopping intensity (from Low to Max) did not have a significant effect on Sol fascicle behavior but increased the initial shortening of the GaM muscle fascicles (P<.001). Interestingly, the lengthening of GaM fascicles tended to begin later with increasing hopping intensity (Fig. 12).

Fig. 13 emphasizes further the differences in fascicle behavior between Sol and GaM in hopping. As expected, the tendon force increases with increasing hopping intensity for both muscles. However, clear differences were observed in the timing of the peak tendon force and length changes of fascicles between the muscles. For the Sol, peak force occurs close to the end of fascicle lengthening but for the GaM the peak tendon force is reached before the end of fascicle lengthening at Med and Max hopping. In addition, the operating length of Sol fascicles was found to remain unchanged, whereas GaM fascicles

38

demonstrated a clear shift towards shorter fascicle lengths with increasing hopping intensity (Fig. 13).
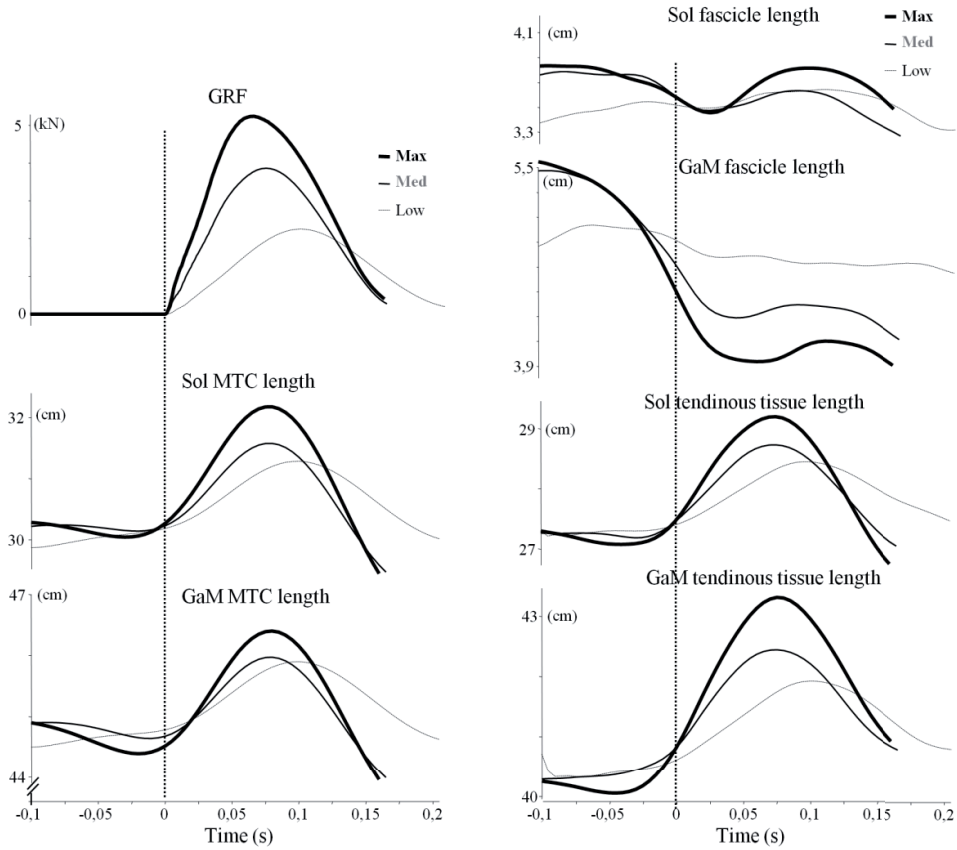
FIGURE 12   Vertical ground reaction force (GRF) and the length of muscle-tendon complex (MTC), fascicles and tendinous tissue of soleus (Sol) and gastrocnemius medialis (GaM) muscles in hopping at different intensities. Data represent means of the subjects. The vertical line represents the beginning of the ground contact.
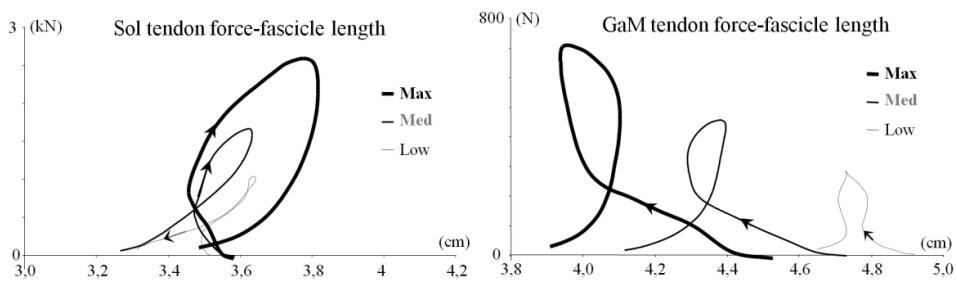
FIGURE 13   Fascicle lengths of the soleus (Sol) and gastrocnemius medialis (GaM) muscles plotted against the calculated tendon forces of the muscles in hopping at different intensities. Data represent means of the subjects. The arrowheads indicate the direction of the force-length curves.

Despite the observed differences in fascicle behavior between Sol and GaM, the tendinous tissue behaved in a similar manner in these muscles and followed the lengthening-shortening behavior of the MTC during the ground contact phase in hopping (Fig 12, lower panels). The amplitude of both lengthening and shortening increased significantly with increasing hopping intensity. It can also be seen from Fig. 12 that the length changes of the whole MTC is mainly taken up by the tendinous tissue. This could imply that the fascicles could operate within a relatively narrow length range.

5.1.2 Pre-activation and stretch reflex activation

EMG activity prior to and during the ground contact phase increased gradually with increasing hopping intensity (Fig. 9). This increase was significant for all the measured muscles for pre-activation and BGA. Pre-activation of Sol and GaL correlated significantly to ankle joint stiffness ($r = .47$, P<.01 and $r = .33$, P<.05, respectively) and leg stiffness ($r = .41$, P<.05 and $r = .48$, P<.01, respectively) for the entire data pool. Interestingly, GaM PRE EMG was found to be significantly related only to knee stiffness ($r = .47$, P < .01). Pre-activation of GaM was also significantly related to the initial fascicle shortening during the early ground contact phase (Fig. 14).
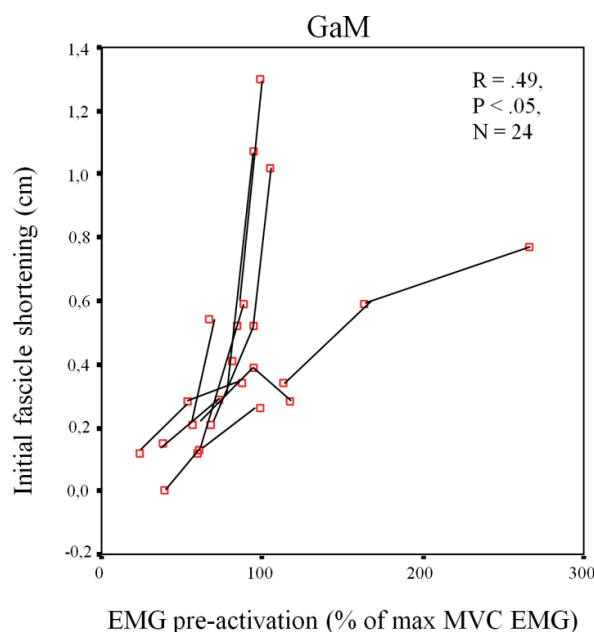
FIGURE 14   Relationship between the initial fascicle shortening and pre-activation EMG (normalized to the maximal EMG in the maximal isometric voluntary plantarflexion test, MVC EMG) for gastrocnemius medialis (GaM) muscle in hopping. Data represents the individual values of eight subjects from all the hopping intensities.

Significant correlation between the pre-activation and the initial fascicle shortening was also found in Sol for the entire data pool but within individuals it was not consistent as in GaM.

Despite the increased pre-activation and BGA, the short-latency stretch reflex response increased only in VL muscle with increasing hopping intensity. However, no significant relationships were found between M1 EMG and stiffness parameters for any of the measured muscles.

5.1.3 Interaction with jumping performance

No significant relationship was found between the leg or ankle joint stiffness and the take-off velocity (P=.23 and P=.12, respectively). However, within the entire data set (n = 36) the knee joint stiffness was significantly correlated to the take-off velocity (R = .56, P<.001). Leg stiffness was found to be significantly related to the ankle joint stiffness at all the intensity levels in hopping (R = .72 - .92, P<.05 - .01) and within the entire data set this relationship was best described with a logarithmic fit (R = .87, P<.001). No significant relationship was observed between knee joint and leg stiffness. However, knee joint stiffness was significantly correlated both to touch-down (r = -.75, P<.001) and minimum knee angles (r = -.58, P<.001).

5.2 Effects of exhausting SSC exercise on leg and joint stiffness regulation

5.2.1 Changes during the SSC exercise

5.2.1.1 Kinetic and kinematic parameters

Fig. 15 shows an example of force and EMG responses during the submaximal continuous jumping until exhaustion. No changes were observed in the selected parameters during the preceding 100 maximal intermittent drop jumps, although large inter-individual variation existed (Fig. 16). Different from the maximal jumps, the submaximal continuous jumping at 70% intensity until exhaustion induced a significant reduction in leg stiffness and other mechanical parameters. The reduction in leg stiffness during the submaximal jumping was accompanied by a decrease in peak force and increase in contact time. However, no changes were observed in movement amplitude. Individual variation in the measured parameters was found to be large again.
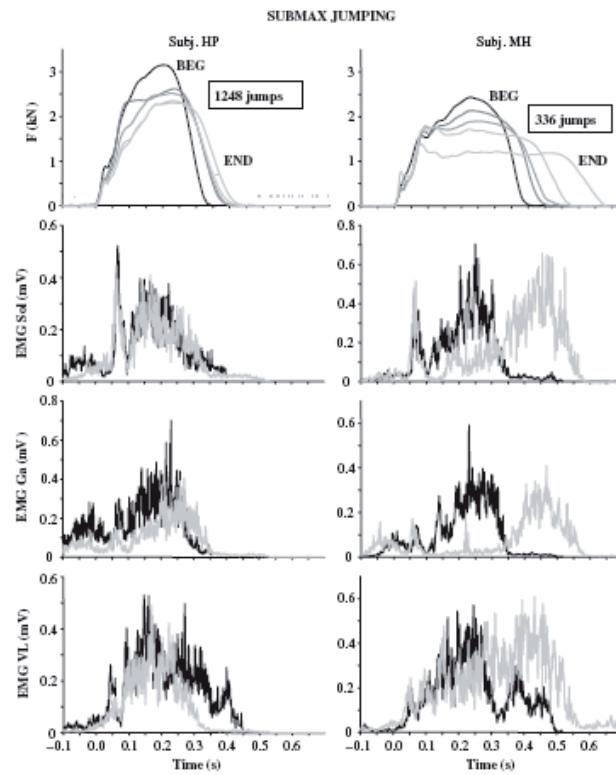
FIGURE 15   Examples of force (F) and rectified EMG changes of the soleus (Sol), gastrocnemius (Ga) and vastus lateralis (VL) muscles of a slow-exhausted (left, 1248 jumps) and a fast-exhausted subject (right, 336 jumps) during the submaximal continuous jumping until exhaustion. Force curves show the gradual increase in contact time with simultaneous decrease in F from the beginning (BEG, black curve, jumps 6-15) to the end (END, light grey curve, last ten jumps) of the submaximal jumping (the intermediate grey curves correspond to the phases of 25%, 50% and 75% of the submaximal jumping). These subjects showed different muscle activation patterns already at BEG. The EMG ratios (braking/push-off phase EMG) of Sol, Ga and VL were 1.44, 1.20 and 1.07 for the subject HP and 0.90, 0.73 and 0.91 for the subject MH, respectively. The EMG curves revealed a shift in activation from the braking to the push-off phase, particularly in the fast-exhausted subject MH (corresponding EMG ratios of 0.38, 0.24 and 0.79 at END).
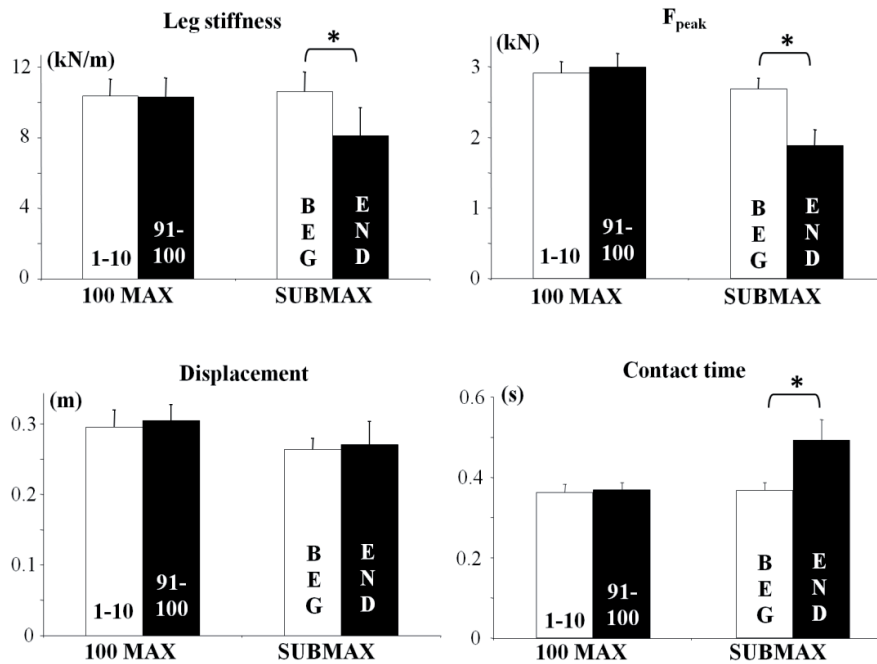
FIGURE 16   Group mean values (n=8) of leg stiffness, peak force (F_{peak}), movement amplitude (sledge seat displacement during the braking phase, Displacement) and contact time of the first (1-10) and last (91-100) ten of the 100 maximal intermittent drop jumps (100 MAX) as well as at the beginning (BEG, jumps 6-15) and end (END, last ten jumps) of the following submaximal continuous jumping until exhaustion (SUBMAX). Values represent means (+ S.E.M.). *, significantly different (P<.05)

5.2.1.2 Muscle activation

Similarly to the changes in mechanical parameters, muscle activation showed no change during the maximal intermittent jumps and a significant decrease during the submaximal continuous jumping (Fig. 17). The aEMG during the pre-activation and the contact phase decreased significantly in Sol and GaM muscles. Similar trend was also observed in VL muscle but the changes were not statistically significant. High EMG activity in braking phase relative to push-off phase (EMG ratio) was significantly related to leg stiffness at the end of submaximal jumping (Fig. 18).
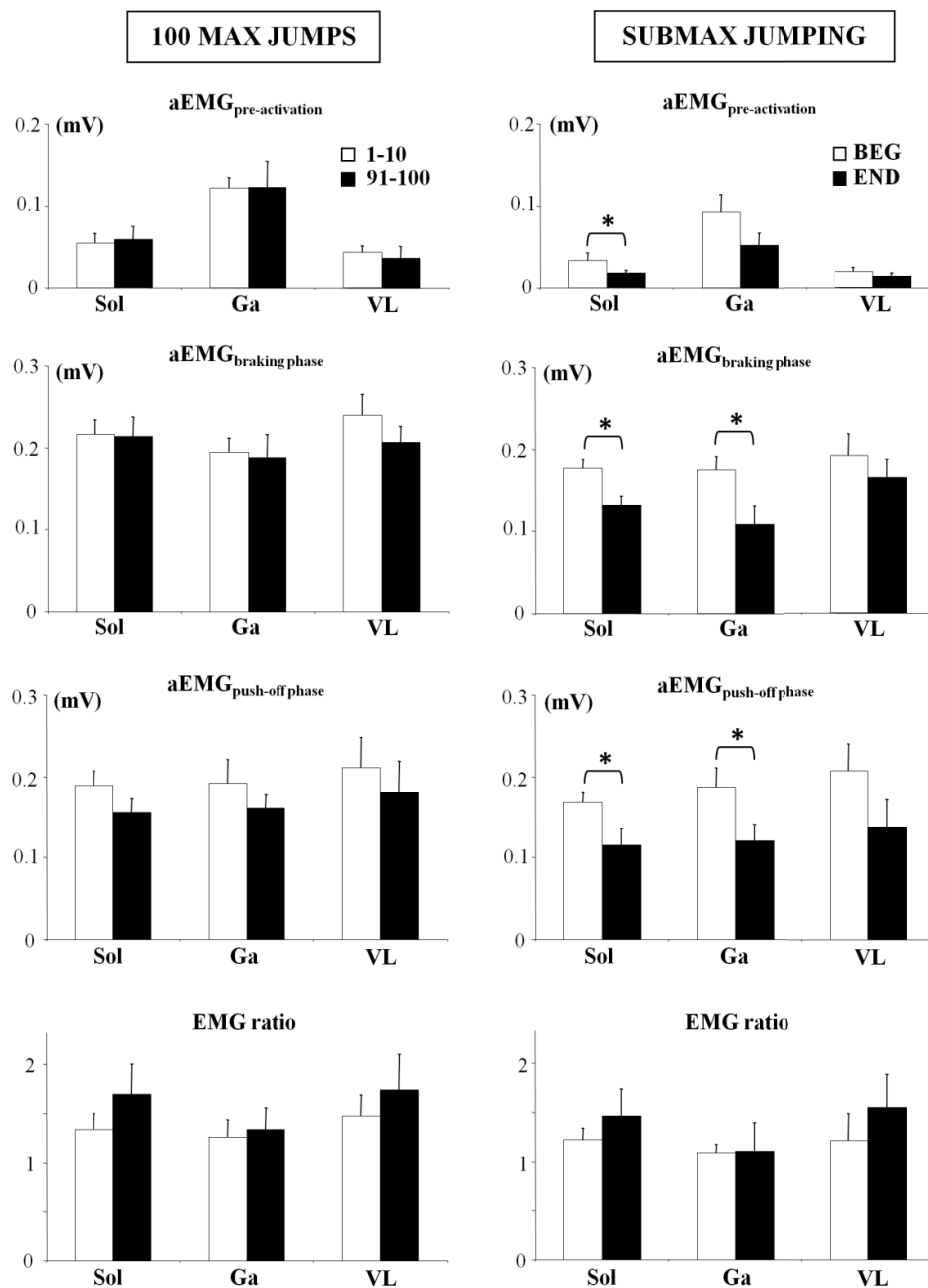
FIGURE 17 Group mean values (n=8) of averaged EMG (aEMG) of the soleus (Sol), gastrocnemius (Ga) and vastus lateralis (VL) muscles in the pre-activation phase, braking phase and push-off phase of the first (1-10, open bars) and last (91-100, filled bars) ten of the 100 maximal intermittent drop jumps (on the left) as well as at the beginning (BEG, jumps 6-15, open bars) and end (END, last ten jumps, filled bars) of the following submaximal continuous jumping until exhaustion (SUBMAX). The lowest graphs represent the corresponding values for EMG ratios (braking/push-off phases). Values are means (+ S.E.M.). *, significantly different (P<.05)

SUBMAX JUMPING (END)
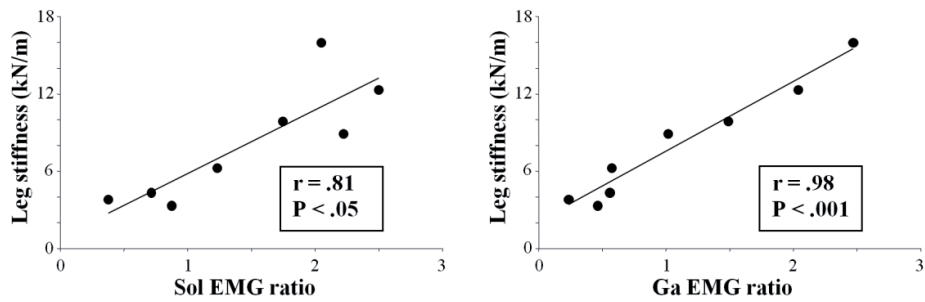
FIGURE 18   Relationship between leg stiffness and EMG ratio (braking/push-off phase) of the soleus (Sol) and gastrocnemius (Ga) muscles at the end of the submaximal jumping.

5.2.2 Acute and long-term changes post-exercise

The SSC exercise protocols used in the present study induced long-lasting impairments in muscle performance, with some of the measured parameters in a bimodal fashion (Fig. 19) (e.g. Nicol et al. 2006). This bimodality was mainly seen in EMG responses.
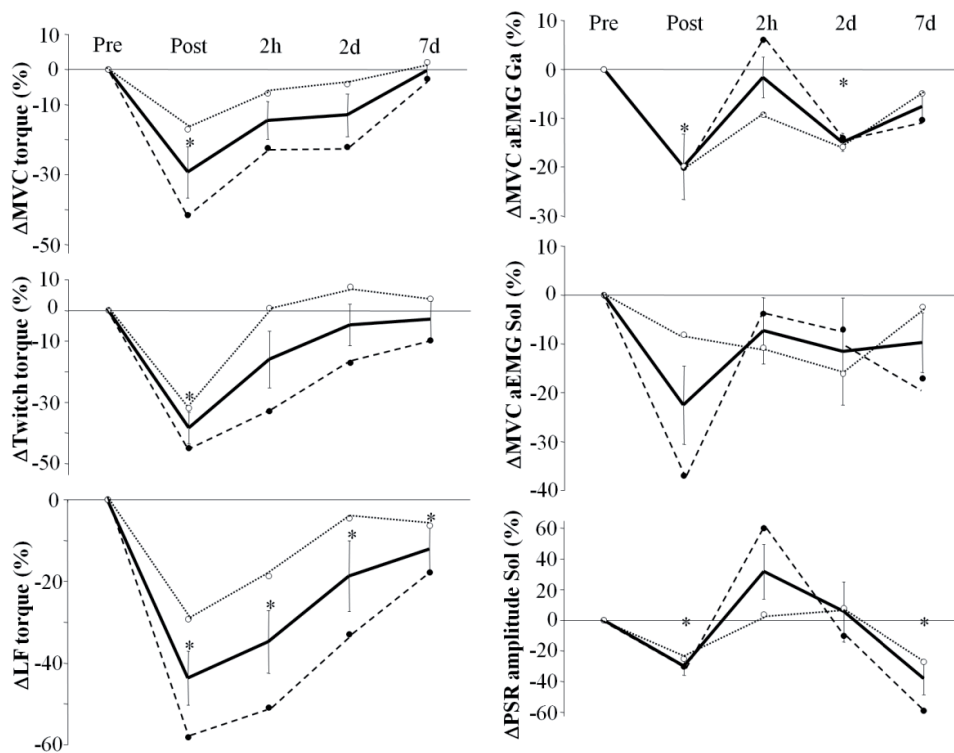
FIGURE 19   Exercise-induced changes (mean ± S.E.M.) in plantarflexor torque in maximal isometric voluntary contraction (MVC), maximal twitch torque, torque during tetanic low-frequency (LF) stimulation, averaged EMG (aEMG) of Ga and Sol muscles during the MVC and peak-to-peak amplitude of passive stretch reflex (PSR) in the Sol muscle. Circles represent the mean values for the subgroups of the fast-exhausted (n=3, open symbols, dotted lines) and slow-exhausted (n=3, filled symbols, dashed lines) subjects. Time scale in horizontal axis represents measurements pre exercise (Pre) and immediately after (Post) as well as two hours (2h), two days (2d) and seven days (7d) post exercise. * Statistically different from Pre value (P<.05)

5.2.2.1 Joint kinetics and kinematics and EMG changes in jumping tests

The present exhausting SSC exercise induced a clear reduction in joint stiffness in rebound jump tests that was found to last several days post-exercise (Fig. 20). The acute reduction in joint stiffness was more prominent in knee than in ankle. However, the recovery of joint stiffness to pre-exercise level tended to take longer in ankle joint. The post-exercise changes in joint stiffness followed well the changes in joint angle, whereas in ankle joint it seemed to be more sensitive to changes in joint moment. Different from the mechanical parameters, the EMG activity showed a rapid recovery within 2 hours after the SSC exercise, which was followed by a secondary decline in some cases. Exercise-induced changes in pre-activation of both triceps surae and VL muscles were significantly correlated to changes in joint stiffness (Fig. 21) (I).
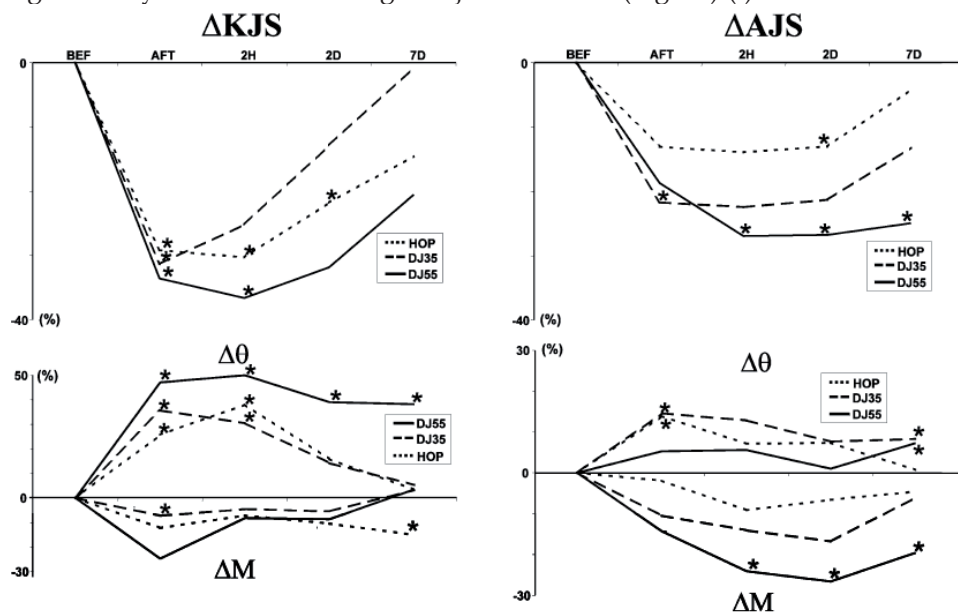
FIGURE 20   Relative changes in knee (KSJ; upper left panel) and ankle joint stiffness (ASJ; upper right panel) in different jumping tests (HOP, DJ35, DJ55). Lower panels represent the relative changes in joint angles (Δθ) and joint moments (ΔM) for the knee (left panel) and ankle (right panel). Please note the different scales for ankle and knee in the lower panels. Data represent means of the subjects. *, significantly different from the pre-exercise value.
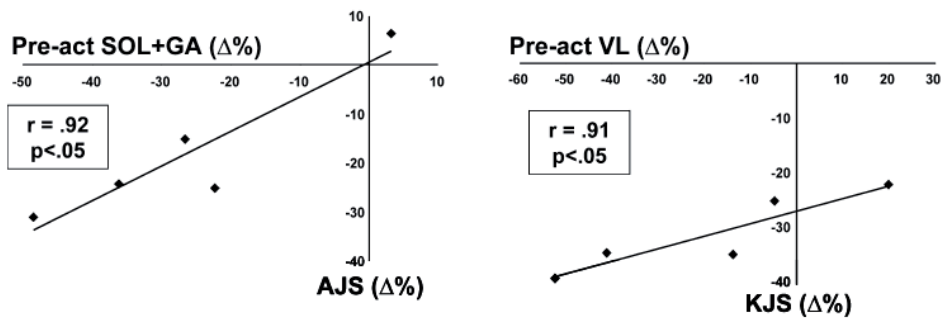
FIGURE 21   Left: Relationship between changes in triceps surae (SOL+GA) preactivity and ankle joint stiffness (AJS) immediately after the fatiguing SSC exercise. Right: The corresponding relationship between changes in VL preactivity and knee joint stiffness (KJS). Points represent individual mean changes of all jumping tests (HOP, DJ35 and DJ55).

5.2.2.2 Neuromuscular properties of MTC in isometric tests

Our results revealed significant post-exercise reduction in muscle force production characteristics, both in MVC and electrically evoked contractions (Fig. 19) (I, III) as well as mechanical properties of MTC (Fig. 22) (II). Recovery of muscle force production was slow, and for some parameters incomplete still 7 days post-exercise, similarly to joint stiffness.
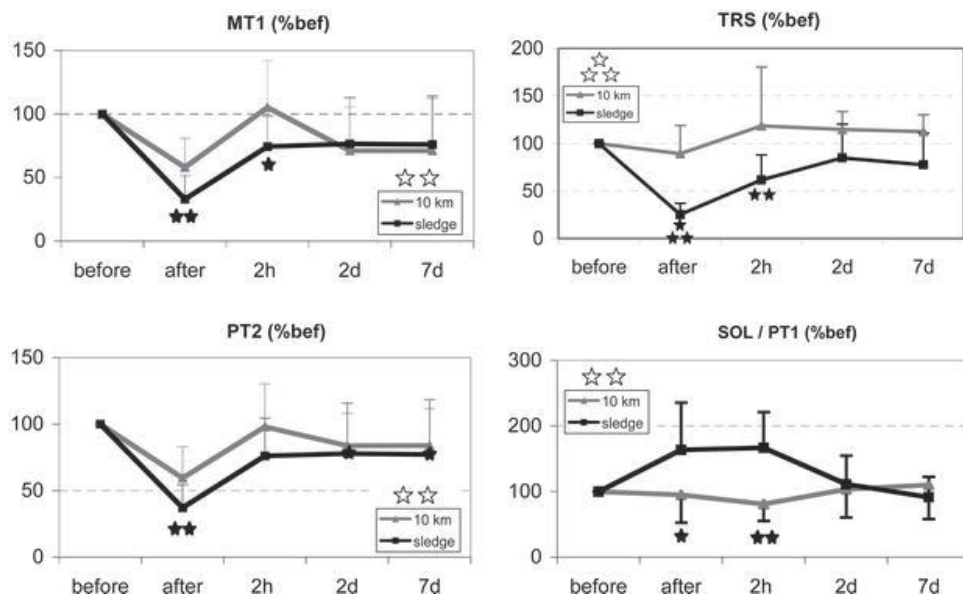
FIGURE 22   Acute and delayed fatigue effects on the mechanical and EMG reflex responses in the 10 km and sledge subgroups. Data represent means of the subjects. The changes are expressed as a percentage of the before fatigue values. Mean torque (*MT1*), double stretch-induced peak torque (*PT2*), twitch resistance to stretch (*TRS*) and SOL EMG/peak torque (*SOL/PT1*) ratio. $^*P<0.05$, $^{**}P<0.01$ and $^{***}p<0.001$, inter-exercise difference on a given day; *open stars,* interaction Subgroup × Testing day

Muscle activity during MVC showed immediate post-exercise decline, similarly to force parameters (Fig. 19) (I, III). Different from the slow recovery of force, MVC EMG showed a rapid recovery and a secondary decline 2 days post-exercise. Similar bimodal recovery pattern was observed in PSR EMG response (Fig. 19) (II, III).

5.2.2.3 Metabolic responses

Fig. 23 shows the post-exercise B-La and CK responses after the exhaustive jumping SSC exercise. As expected, significantly higher B-La values were observed after intensive jumping than after 10-km running exercise (II). However, CK activity showed a similar trend after the both exercise types with a delayed peak value 2-7 days post-exercise. Peak B-La at the end of submaximal jumping exercise was significantly correlated to corresponding leg stiffness, i.e. the higher the B-La the lower the leg stiffness (Fig. 24) and EMG ratio of Sol and Ga muscles (Fig. 25), respectively (IV).
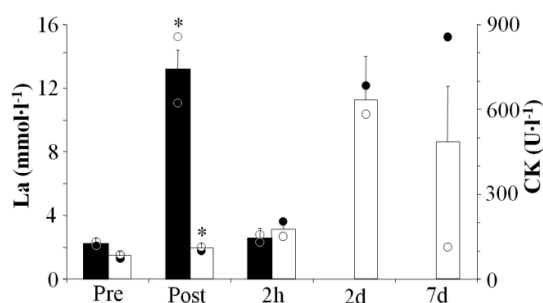
FIGURE 23   Exercise-induced changes (mean ± S.E.M.) in blood lactate (filled columns) and serum creatine kinase (open columns). Circles represent the mean values of the fast-exhausted (n=3, open symbols) and slow-exhausted (n=3, filled symbols) subjects. Time scale in horizontal axis represents measurements pre exercise (Pre) and immediately after (Post) as well as two hours (2h), two days (2d) and seven days (7d) post exercise. * Statistically different from Pre value (P<.05)
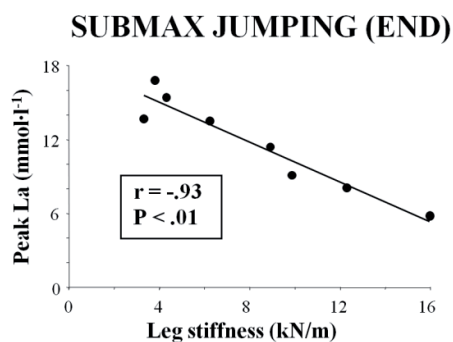
FIGURE 24   Relationship between the post exercise peak lactate (Peak La) concentration and leg stiffness at the end of the submaximal jumping.

48

SUBMAX JUMPING (END)
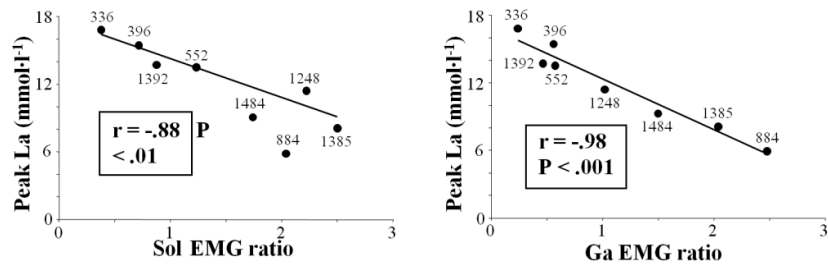
FIGURE 25   Relationship between the post exercise peak lactate (Peak La) concentration and EMG ratio of soleus (Sol) and gastrocnemius (Ga) muscles at the end of the submaximal jumping. The numbers next to the points indicate the number of jumps performed in the submaximal continuous jumping until exhaustion.

5.2.3 Individuality of recovery

The present results indicated great individuality in the recovery pattern from the exhaustive SSC exercise, regardless of the exercise mode (jumping, running). In fact, two types of recovery patterns, fast- (FR) and slow-recovery (SR), were identified according to the neuromuscular and metabolic responses to the SSC exercises used. FR subjects were characterized by a quick recovery of muscle force production (Fig. 19, left panels) and a faster and less prominent kinetics of CK activity. Our data indicate that the different recovery profiles may be related to the total mechanical loading and/or duration of the exercise. The greater total number of jumps during the SSC exercise was found to be related to greater decreases in voluntary activation and muscle force production ($r = .94$, $P<.01$, for both).

6   DISCUSSION

The main observations of the present series of studies led to the following hypotheses and/or argumentations:

1) When the SSC exercise is performed with a short contact with the ground, the leg stiffness is mainly adjusted by modulating ankle joint stiffness. The role of knee joint stiffness is more related to the level of hopping intensity. Centrally pre-programmed motor commands play major roles in leg stiffness adjustments during SSC exercise.

2) Behavior of muscle fascicles differed between Sol and GaM muscles in short-contact SSC exercise. The operating length of GaM fascicles shifted towards shorter fascicle lengths in GaM, but not in Sol, with increasing hopping intensity. Furthermore, the lengthening of fascicles during the ground contact occurred later in GaM than in Sol. This may indicate differential roles of these muscles in joint and leg stiffness modulation.

3) Exhaustive SSC exercise induced reduction in muscular force output and its recovery took place in a bimodal fashion. The SSC exercise caused reduction also in ankle and knee joint stiffness that lasted several days after the exercise.

4) The fatigue-induced changes in joint stiffness followed the changes in joint angular displacement for the knee, whereas the changes in ankle joint stiffness seem to be more sensitive to changes in joint moment. While the recovery of muscle force output demonstrated a bimodal pattern the stiffness parameters did not always followed this model.

5) The immediate post-exercise reduction in both muscle performance and joint stiffness after exhaustive SSC exercise are likely related to both central and peripheral (metabolic) fatigue, whereas the long-term impairments most likely derive from exercise-induced muscle damage.

6) EMG activity was recorded primarily from the main knee extensor and plantarflexor muscles. From these muscles, the triceps surae muscle group, particularly GaM, played an important role in leg stiffness modulation during exhaustive SSC exercise. In addition to the important role of GaM preactivation in leg stiffness adjustments, it was found that high EMG ratio (high muscle activation during the braking phase relative to the push-off phase) is a prerequisite for efficient stiffness modulation during exhaustive SSC exercise. This is likely favouring the storage and utilization of elastic energy, which may contribute to SSC performance by delaying the exhaustion (termination of exercise).

6.1 The role of knee and ankle joint stiffness adjustments in leg stiffness modulation during SSC exercises

6.1.1 Effects of exercise intensity

Leg stiffness is likely adjusted according to the task and intensity requirements of SSC exercise. In the present study (V), leg stiffness was found to be fairly constant throughout wide range of hopping intensities. On the other hand, Farley and Morgenroth (1999) found that leg stiffness was twice as great for maximum height hopping as for preferred height hopping at fixed hopping frequency (2.2 Hz) and freely chosen contact times. Shortening the ground contact time (by instructing the subjects to jump with shorter contact time) has been shown to increase leg stiffness during drop jumps (Arampatzis et al. 2001) and hopping (Hobara et al. 2007) with maximal effort. In the present study, the subjects were asked to jump with a shortest possible contact time (but freely chosen frequency) at all hopping intensities. In a study of Hobara et al. (2008), the subjects were asked to perform hopping at two different frequencies, low (1.5 Hz) and high (3.0 Hz), which match well the observed hopping frequencies for high (1.6 Hz) and low (2.9 Hz) hopping intensities in the present study. They also used a similar hopping to the present study (with shortest possible contact time). However, they found greater leg stiffness with high than low frequency hopping does differ to the present finding of constant leg stiffness. The possible mechanisms for leg stiffness regulation during short-contact SSC exercises are discussed as follows.

Ankle and knee joints seem to have different roles in stiffness regulation in hopping (V). The constant ankle joint stiffness with increasing hopping intensity together with significant correlation to leg stiffness suggest that ankle

joint stiffness is tightly coupled with leg stiffness modulation. At the same time, the increase in the knee joint stiffness with the hopping intensity indicates that knee joint stiffness plays an important role for optimizing the force/power output in short-contact SSC exercise (Horita et al. 2002), rather than modulating leg stiffness. To explain the observed sensitivity of leg stiffness to changes in ankle joint stiffness in SSC exercise, two possible mechanisms are offered. First, in a system with multiple springs in series, the least stiff spring undergoes greatest displacement for a given force and therefore, has the greatest influence on the overall stiffness of the system. Ankle joint was found to be less stiff than knee joint in the present study (V) thus favouring the least stiff spring theory. Although hip joint stiffness was not measured in the present study, the earlier studies using similar hopping task to the present study indicate a trend for higher hip than ankle joint stiffness (Hobara et al. 2008, 2010). Secondly, the moment arm of GRF is largest at the ankle resulting in a larger moment and angular displacement in ankle than in the knee or hip joint (Farley et al. 1998). Furthermore, rotation of foot segment has greater effect on vertical displacement of center of mass, as compared to the other leg segments, due to its more horizontal position. As ankle joint stiffness has been shown to play an important role in leg stiffness adjustments (Arampatzis et al. 2001; Farley et al. 1998; Farley and Morgenroth 1999), the following discussion focuses on the tendomuscular factors likely to contribute to ankle and, consequently, leg stiffness regulation.

6.1.1.1 Muscle stiffness regulation during SSC exercise

It is evident from the literature that the interaction between contractile and elastic components of MTC during human movements is highly specific, depending e.g. on the task / type of contraction, force-length relation, intensity and muscles involved (for reviews, see Ishikawa and Komi 2008; Kawakami and Fukunaga 2006). In line with this, our data revealed differential behaviour between Sol and GaM muscles in hopping, which may indicate different roles of mono- and biarticular muscles during SSC muscle action (Ishikawa et al. 2005b; Sousa et al. 2007). Sol fascicles were found to operate within a narrow length range that was independent of the hopping intensity, whereas the operating range of GaM fascicles shifted towards shorter fascicle lengths with increasing hopping intensity. It is not clear if this shift to shorter fascicle lengths occurs only to enhance the stretch and recoil of tendinous tissue or if it is also related to other functions, e.g. coupling of joint movements and/or energy transport between the joints (e.g. van Ingen Schenau et al. 1990).

Furthermore, the observation that the fascicle lengthening during the ground contact tended to occur later with increasing hopping intensity in GaM, but not in Sol, indicate that stretch reflexes may have different function in stiffness regulation during SSC locomotion in mono- and biarticular muscles. While the stretch reflexes in Sol are likely to contribute to muscle force already in the braking phase in hopping, for the GaM muscle this effect may take place during the the push-off phase. Similar observation was also made by Ishikawa

and Komi (2007) in running. This may indicate that in short-contact SSC muscle actions, such as running and hopping, stretch reflex functions to increase the muscle force during the braking phase in Sol, whereas in GaM the mechanical outcome contributes to subsequent push-off action. It may also provide further explanation for the less prominent stretch reflex response observed in GaM than in Sol muscle during SSC muscle actions (Funase et al. 2001; Maton and Le Pellec 2001; Voigt et al. 1998).

The observed small length changes of Sol and GaM fascicles, as compared to the corresponding length changes of tendinous tissues, refer to high muscle stiffness and efficient tendon recoil characteristics in hopping. In fact, the fascicles of Sol and GaM were found to shorten at the beginning of the ground contact due to high preactivation of the muscles. This shortening of fascicles enables efficient usage of tendon elasticity, since the simultaneous lengthening of the whole MTC is thus taking up by the tendinous structures. Interestingly, only GaM fascicles were found to be affected by hopping intensity. The observed shortening of GaM fascicles during the ground contact in hopping would also be advantageous for coupling of joint movements between ankle and knee due to tendinous action (e.g. van Ingen Schenau et al. 1990). This mechanism could offer one explanation for the finding that GaM was found to play an important role in leg stiffness regulation during fatiguing SSC exercise (IV).

6.1.2 Effects of fatigue

Exhaustive SSC exercise induces acute and long-term impairment in leg stiffness regulation. The present results suggest that ankle joint plays a key role in leg stiffness adjustments during SSC fatigue (I, IV). To support our hypothesis, inducing fatigue in knee extensor muscles (with minimal or no loading on ankle extensor musculature) had no influence on post-exercise leg stiffness in SSC jumping test (Harrison and Gaffney 2004; Padua et al. 2006). However, clear reduction in leg stiffness has been observed during a rebound jumping exercise (IV) and running (Dutto and Smith 2002), both involving significant contribution of the ankle joint. Therefore, it seems that during fatiguing SSC exercise the leg stiffness is affected most strongly if, besides the changes in the knee joint, the ankle joint is also experiencing impairments of its own stiffness regulation.

Earlier studies have reported a prolonged impairment of knee joint stiffness after exhausting SSC exercise (Horita et al. 1996 1999). Our results are in line with the previous findings exhibiting reduced joint stiffness still 7 days post-exercise (I). There was a tendency towards greater decrement and slower recovery of ankle joint stiffness with greater loading demands of SSC task (submaximal hopping vs. maximal drop jumps). This is supporting the hypothesis of task-dependent effects of SSC fatigue (Nicol et al. 2006). Therefore, our results suggest that adaptation mechanisms of stiffness regulation are truly different between knee and ankle joints. The observed post-exercise changes in knee joint stiffness followed well the corresponding changes

in knee angular displacement, whereas the changes in ankle joint stiffness were more related to changes in ankle joint moment. The post-exercise increase in knee angular displacement may indicate a compensatory mechanism to reduced ankle force output due to fatigue, as has been reported in fatiguing hopping task (Bonnard et al. 1994). Similar compensatory action was also found by Horita (2000) in relation to fatiguing rebound jump exercise. He observed decreased leg stiffness during the fatiguing SSC exercise that was accompanied by increased knee extensor activity and angular displacement of the knee joint. The fact that in the present study knee extensor activity showed a tendency to decrease, rather than increase, during the SSC exercise is likely related to the present more exhaustive SSC protocol (until complete exhaustion) than in the study of Horita (2000), who used 'task failure' (e.g. Gandevia 2001) as cessation of exercise. The neuromuscular mechanisms underlying impaired post-exercise ankle joint stiffness have been addressed in the following chapter.

6.2 Contribution of central and peripheral mechanisms to joint and leg stiffness regulation during SSC exercise and recovery

6.2.1 Central and peripheral mechanisms related to SSC fatigue

As brought up in a review of Nicol et al. (2006), SSC fatigue is a complex phenomenon involving various neural, metabolic and mechanical adaptation mechanisms that may vary depending e.g. on the SSC task itself. Characteristic to SSC fatigue is a prolonged recovery, lasting more than one week in some cases, which often follows a bimodal trend (Nicol et al. 2006, Dousset et al. 2007). This chapter focuses on the central and peripheral mechanisms contributing to impaired force/power output and consequently to stiffness regulation during SSC exercise.

6.2.1.1 Central fatigue

Central fatigue, i.e. failure to activate the muscles voluntarily (for a review, see Gandevia 2001), in SSC muscle actions is often related to long-term SSC exercises (Millet et al. 2002, 2003; Place et al. 2004). On the contrary, an increase (potentiation) in muscle activation has been observed after intensive short-term SSC exercise both in isometric MVC (Strojnik and Komi 1998) and maximal SSC task (Horita et al. 1996, 1999). In the present SSC exercise (modified from the experiment of Horita et al. 1996; Nicol et al. 1996) the SSC task was extended until complete exhaustion in order to explore if central fatigue can be induced by intensive short-term SSC exercise.

Differering from the previous intensive short-term SSC exercises (Horita et al. 1996, 1999; Strojnik and Komi 1998), the present study indicates that central fatigue may also contribute to impaired performance after intensive short-term

SSC exercise (I, III). This activation failure was, however, related to total number of jumps during the SSC exercise suggesting that central fatigue may be related to the exercise duration and/or volume (III). This is in line with the observations of Bigland-Ritchie et al. (1986) regarding intermittent submaximal isometric exercise. Furthermore, it is likely that the SSC fatigue model used needs to be totally exhaustive (beyond the endurance limit) in order to induce central fatigue during short-term intensive SSC exercise. The previous intensive short-term exercises (that were stopped when the pre-determined submaximal work level was not maintained anymore) showed an increase (potentiation) in muscle activation after intensive short-term SSC exercise both in isometric MVC (Strojnik and Komi 1998) and maximal SSC task (Horita et al. 1996, 1999). This indicates a central compensation mechanism to counteract the peripheral failure in muscle performance.

The observed central fatigue could be at least partly of peripheral origin via reflex loops (Garland 1991). Metabolic by-products of intense muscular exercise are known to sensitize group III and IV muscle afferents (Mense and Meyer 1988; Rotto and Kaufmann 1988) and may lead to presynaptic inhibition of the a-motoneuron pool (Duchateau and Hainaut 1993; Garland 1991). This mechanism has been suggested to play a role in post exercise reduction of reflex sensitivity in fatiguing SSC exercise (Avela et al. 1999; Dousset et al. 2007; Nicol et al. 1996). The observed post exercise reduction in the PSR (and a similar trend in the H-reflex response) supports this theory. However, contribution of other mechanisms for the declined activation level, such as disfacilitation of α-motoneuron pool (Macefield et al. 1991) and supraspinal fatigue (e.g., Gandevia 2001), cannot be excluded either. In fact, supraspinal fatigue has been suggested to occur after various sports activities (Hollge et al. 1997; Ross et al. 2007; Verin et al. 2004).

6.2.1.2 Peripheral fatigue

The significant post-exercise force reduction in electrically evoked muscle contractions of triceps surae muscle refers to peripheral (contractile) failure after the present SSC exercise (I, III). This is similar to earlier SSC studies using both short-term (Skurvydas et al. 2000; Strojnik and Komi 1998) and prolonged SSC fatigue models (Millet et al. 2003). Metabolic fatigue (accumulation of metabolites, e.g. B-La) is likely to play a role in contractile failure immediately after intensive SSC exercise by impairing the excitation-contraction coupling (Hill et al. 2001; Li et al. 2002). The significant high correlation between the peak post-exercise B-La and the leg stiffness at the end of the exercise (IV) gives further support to metabolic fatigue as an important factor on leg stiffness regulation through impaired force production ability.

It must be noted that neural control may play an important role in development of metabolic fatigue during intensive SSC exercise. High EMG activity of plantarflexor muscles during the braking phase of ground contact (relative to the push-off phase; EMG ratio) was found to be significantly correlated to B-La at the end of the exhaustive SSC exercise, i.e. the higher the

EMG ratio the lower the B-La (IV). Possible explanation for this relationship is that high braking phase activation favors the storage (and utilization) of elastic energy and thus, less muscular work (and usage of metabolic energy) through active muscle contraction is needed during the push-off phase. Dalleau et al. (1998) reported lower energy cost of running with greater leg stiffness, indicating better utilization of elastic energy with higher stiffness. The present findings further suggest that the high leg stiffness through the high EMG ratio may prevent or attenuate the development of metabolic fatigue and consequently, delay the time to exhaustion during fatiguing SSC exercise. Supporting this hypothesis, the subjects with higher EMG ratio (and lower B-La) at the end of the exhaustive SSC exercise were the ones with greater total number of jumps. Fig. 26 shows the EMG ratios of one subject during the exhaustive submaximal jumping exercise. The sudden decline in EMG ratios at the end of the exercise was accompanied by high post-exercise B-La that was different from the other subjects with high number of jumps during the exercise. This may refer to metabolic fatigue as a cause of exhaustion for this particular subject.
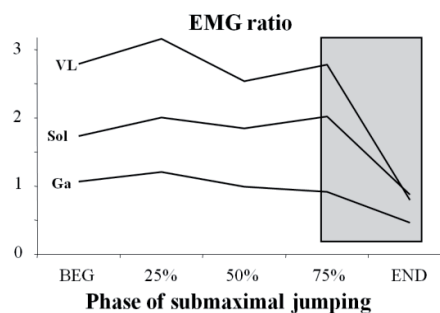
FIGURE 26   EMG ratios (braking/push-off phases) of the soleus (Sol), gastrocnemius (Ga) and vastus lateralis (VL) muscles of one subject during the different phases of submaximal continuous jumping. Note the sudden decline in the EMG ratios during the final phase of the submaximal jumping (shaded area).

6.2.1.3 Exercise-induced muscle damage

On the other hand, the recovery of metabolic fatigue was fast (indicated by B-La kinetics), whereas the electrically evoked (low-frequency stimulation) force exhibited a slow recovery that was incomplete still one week after the SSC exercise (I, III). This slow recovery of LF force indicates that exercise-induced muscle damage contributed to the present contractile failure (Edwards et al. 1977; Yeung et al. 2002). This theory is further supported by the observed elevated CK activity after the SSC exercise that has been used as an indirect marker of muscle damage (for a review see Warren et al. 1999).

Our results also revealed a great inter-individual variation in the post-exercise responses in the measured parameters, particularly in plantarflexor

force (I, II, III). In fact, the subjects exhibited two distinct subgroups according to their neuromuscular responses to the SSC exercises, regardless of the exercise type (II, III). The fast-recovery subjects showed a quick recovery of force within two days post-exercise, whereas the slow-recovery subjects demonstrated a delayed force recovery and elevated CK activity lasting more than one week in some individuals. It is possible that the exercise-induced muscle damage and subsequent inflammation was more severe in slow-exhausted subjects and thus, delayed the recovery as compared to the fast-recovery subjects. Interestingly, we found a significant correlation between the number of jumps performed in the SSC exercise and the decrease in plantarflexor low-frequency torque as well as in CK activity seven days post-exercise. This implies that SSC exercise volume / duration may play a role in the magnitude of muscle damage. To support this notion, Nosaka et al. (2002) reported greater MVC force reduction and slower recovery after 24 than 12 eccentric elbow flexions. Furthermore, Hesselink et al. (1996) found greater muscle damage and force loss with increasing number of eccentric contractions in rats.

Possible muscle damage and subsequent inflammation may also have an indirect effect on the prolonged impairment in muscle force production through reduced reflex sensitivity. Similar to metabolic fatigue, group III and IV muscle afferents are sensitive to intramuscular increase in temperature, pressure and metabolic by-products related to muscle damage (Kaufmann and Rybicki 1987; Kniffki et al. 1978; Mense and Meyer 1988). The observed relationship between the secondary decline in PSR and CK activity indicates the contribution of muscle damage to the reduced reflex sensitivity. This is in line with the observations of Nicol et al. (1996) and Dousset et al. (2007) using a similar SSC exercise to the present study. However, it has also been suggested that the observed exercise-induced disruption of cytoskeleton is likely due to remodeling of myofibrillar structure rather than a sign of muscle fiber injury (e.g. Malm 2001; Yu et al. 2003).

6.2.2 Neural control of leg stiffness during SSC exercise

Previous studies have emphasized the importance of pre-activation and stretch reflex behavior in stiffness regulation during SSC muscle actions (Arampatzis et al. 2001; Avela and Komi 1998; Dyhre-Poulsen et al. 1991; Gollhofer et al. 1984, 1992; Gollhofer and Kyröläinen 1991; Hobara et al. 2007; Horita et al. 1996, 2002; Moritani et al. 1991). The present study demonstrated a direct relationship between pre-activation and joint / leg stiffness both in fatiguing (I, IV) and non-fatiguing (V) SSC exercises, indicating a strong reliance of centrally programmed motor control in stiffness regulation. The present data also clearly supports the observation that stretch reflexes can have a significant contribution to muscle activity during the braking phase of the ground contact in SSC exercises, as observed earlier (for a review, see Komi and Gollhofer 1997).

The present data indicate muscle-specific differences in leg stiffness regulation during SSC tasks. Moritani et al. (1991) observed greater pre-activation in Sol than in GaM muscles in hopping suggesting a preferential

activation of fast-twitch GaM muscles in this type of short-contact SSC task with high force and power demands. Similar pattern was also found in the present study (IV, V). The purpose of the observed high pre-activation and the fairly constant muscle activity during the contact phase in Ga muscle is likely to maintain the mechanical properties (namely stiffness) of the Ga MTC constant during the loading (ground contact) phase. Constant stiffness of Ga MTC would favor the energy transfer from knee to ankle joint through the tendinous action (Bobbert et al. 1987) that has been proposed as one of the main functions of bi-articular muscles in multi-joint movements (for review, see van Ingen Schenau et al. 1990). The observed quasi-isometric fascicle behavior of GaM muscle supports the notion of tendinous action.

On the other hand, occurrence of stretch reflex response in the present hopping was more obvious in Sol than Ga muscle (V), as has also been noted earlier (Funase et al. 2001; Maton and Le Pellec 2001; Voigt et al. 1998). Several factors may explain the observed differences in reflex behavior, including fewer muscle spindles and higher reflex threshold in Ga than in Sol as well as slower stretching velocity due to simultaneous knee flexion (Voigt et al. 1998). Interestingly, our observations using real-time ultrasonography indicated lengthening-shortening behavior of Sol fascicles, whereas GaM demonstrated a quasi-isometric / shortening fascicle behavior in hopping. This may imply differential muscle spindle length changes between Sol and GaM muscles that could also partly explain the observed differences in stretch reflex responses. It must be mentioned, however, that short-lived lengthening of GaM fascicles have been observed during the ground contact phase in running and drop jump exercises (Ishikawa and Komi 2007; Sousa et al. 2007). It is possible that the high pre-activation of GaM muscle in the present hopping conditions resulted in high stiffness of muscle fibers and the lengthening of MTC was mainly taken up by the tendinous structures. The EMG recordings of those two experiments (Ishikawa and Komi 2007; Sousa et al. 2007) show clearly lower pre-activation than in the present study, thus indicating the differences in muscle fiber stiffness as a likely explanation to the observed differences in GaM fascicle behavior between the studies. We also observed that the stretch reflex EMG response did not increase with increasing hopping intensity, unlike the pre-activation and BGA. This finding further emphasizes the important role of pre-programmed motor commands in stiffness regulation during short-contact SSC tasks, such as hopping.

6.3 Methodological considerations

Several cautions must be taken into account when interpreting the present results. Firstly, for obtaining a high statistical power for the results the number of subjects in the present experiments was low. However, due to the complex

and laborious methods used in the experiments the number of subjects needed to be compromised.

Secondly, although muscles, tendons and ligaments demonstrate spring-like properties, calculating stiffness (as understood in physics) for biological structures entails several limitations and/or sources of errors as pointed out by Latash and Zatsiorsky (1993). These include, among other things, time-, length- and velocity-dependency of biological structures that differentiate them from the notion of stiffness in physics. Furthermore, the applied models used for describing the MTC and joint behavior are somewhat simplified and, therefore, ignore e.g. possible different material properties and/or behavior within and between the structures of MTC (e.g. differential strain of superficial and deep aponeuroses and outer tendon; Magnusson et al. 2008), individual differences in segment mass and inertial parameters, all the degrees of freedom of joints, etc. Although the real and measured values are likely to differ, depending on the accuracy of the model used, we believe that the observed behavior of muscle fascicles, tendinous structures, MTC and joint is valid in the present experiments.

Thirdly, it may be questioned, why the hip joint stiffness was excluded from the analysis in the present experiments. Although hip stiffness has been reported in some of the earlier reports, its role in leg stiffness adjustments in hopping is quite small, as evidenced by several studies (e.g. Farley et al. 1998; Fukashiro and Komi 1987; Hobara et al. 2009). Furthermore, the phase shift between the peak angular displacement and peak joint moment in the hopping experiment (Experiment 3) was more than 10% in all the hopping conditions. This has been used as an exclusion criterion earlier (Farley et al., 1998) and, therefore, hip joint stiffness was not calculated in that experiment. For the Experiment 1, the fatigue task used was focused to load primarily the ankle and knee extensor muscles and thus, the main focus was put on the stiffness regulation mechanisms of the ankle and knee joints. It must be admitted, however, that hip joint may have played a role in compensating the fatigue effects of knee and ankle musculature on jumping performance and overall leg stiffness adjustments.

One may also criticize the usage of La and CK as the only biochemical markers of muscle fatigue and damage (inflammation). We selected those two markers, since they have been commonly used in earlier SSC fatigue studies for describing the (metabolic) fatigue and muscle damage (e.g. Nicol et al. 2006). It must be mentioned, however, that the assessment of fatigue and recovery in the present study (Experiment 1) consisted of not only the La and CK but several other neuromuscular parameters that were used together to identify the mechanisms of fatigue and recovery. Furthermore, the biochemical markers related to SSC muscle fatigue were studied in more details in our follow-up study of the present experiment (Dousset et al. 2007).

Regarding the neural control mechanisms of stiffness adjustments, it must me noted that the present experiments were limited to examination of short-latency stretch reflex component only. It has been shown that also the medium- and long-latency reflexes are likely to have a significant contribution to muscle

and joint stiffness regulation during human movements (Allum and Mauritz 1984; Horita et al. 1996; Sinkjær et al. 1998). Undoubtedly, they have contributed to the present EMG recordings as well. However, due to the nature of the present SSC tasks the BGA was high in most of the testing conditions and we were not able to clearly identify the medium- or long-latency stretch reflex components in the present experiments and therefore, did not include them in the EMG analysis.

7 PRIMARY FINDINGS AND CONCLUSIONS

The main findings and conclusions of the present work can be summarized as follows:

1) The results of hopping task at different intensity levels suggest that leg and ankle joint stiffness are not affected by intensity of SSC task, whereas knee joint stiffness increases with increasing exercise intensity (V). This observation indicates that leg stiffness is mainly adjusted by modulating ankle joint stiffness, when the SSC exercise is performed with a short contact with the ground. These results support the previous findings regarding the importance of ankle joint stiffness in leg stiffness modulation. The regulation of knee joint stiffness seems to be more related to adjustment of the required mechanical output of the SSC task. Centrally pre-programmed motor commands play major roles in joint and leg stiffness adjustments during SSC exercise.

2) Behavior of muscle fascicles was found to differ between Sol and GaM muscles in hopping supporting the suggested muscle-spesific differences between mono- and bi-articular muscles during SSC muscle actions. The observed shift in operating length of GaM fascicles towards shorter fascicle lengths with increasing hopping intensity, that was not found in Sol, suggest that GaM fascicle behavior is more sensitive to changes in hopping intensity as compared to Sol. Furthermore, the timing of fascicle lengthening during the ground contact was constant in Sol at all hopping intensities but tended to shift later in GaM with increasing hopping intensity. This may indicate different functions of stretch reflex between Sol and GaM in fast-contact SSC muscle actions. It seems that the stretch reflex in Sol enhances muscle force already during the braking phase of ground contact in hopping, whereas in GaM the mechanical output of the stretch reflex likely contributes to the push-off action. The observed length changes of muscle fascicles of Sol and GaM in hopping were small

compared to the length changes of the whole MTC. This indicates high muscle (fiber) stiffness and consequently, efficient elastic recoil characteristics of triceps surae MTC in hopping with short ground contact time. Interestingly, only GaM fascicle behavior was influenced by changes in hopping intensity. These observations, together with the EMG findings (V), suggest that GaM muscle plays an important role in leg stiffness adjustments in hopping.

3) Exhaustive SSC exercise induced reduction in muscular force output neural activation and consequently, also in joint and leg stiffness. These acute impairments in muscle performance likely resulted from contractile failure (peripheral fatigue) due to metabolic fatigue and possible exercise-induced muscle damage and partly from decreased neural drive to muscles (central fatigue). However, the results also revealed large inter-individual variation in neuromuscular responses to SSC exercise. Metabolic fatigue was a likely cause of early cessation of exercise for part of the subjects and consequently, prevented central fatigue to take place due to short duration of the exercise. Furthermore, the magnitude of peripheral and central fatigue was found to be greater with subjects, who performed greater number of jumps during the exercise. These results indicate that the volume and duration of intensive SSC exercise may affect the level of fatigue and time course of recovery (I, II, III, IV)

4) The recovery of muscle activation (both voluntary and reflex parameters) and MVC force demonstrated a bimodal recovery pattern, with partial recovery and subsequent secondary decline, after the exhaustive SSC exercise. On the other hand, evoked muscle force by electrical stimulation did not follow the bimodal recovery pattern showing a gradual and long-lasting recovery over several days. The recovery of joint stiffness showed a similar gradual recovery that was found to be incomplete still one week post exercise, particularly for the ankle joint. This may indicate that the long-lasting post-exercise reduction in joint stiffness may be associated with contractile failure (within the muscle) likely due to exercise-induced muscle damage. (I, II, III, IV)

5) EMG activity was recorded primarily from the main knee extensor and plantarflexor muscles. From these muscles, triceps surae muscle group, particularly GaM, played an important role in leg stiffness modulation during exhaustive SSC exercise. Its high activation prior to and during the braking phase of ground contact is a prerequisite for efficient stiffness modulation, which may contribute to SSC performance by delaying the exhaustion (termination of exercise) (I, IV).

YHTEENVETO

Lihas- ja niveljäykkyyden säätely normaalin sekä väsyttävän venymis-lyhenemissykli –tyyppisen harjoituksen aikana

Lihas- ja niveljäykkyyden säätely on keskeisessä roolissa jokapäiväisessä liik-kumisessa tehden liikkumisesta sujuvaa ja tehokasta hermostollisten säätely-mekanismien ansiosta hyodyntäen lihas- ja jännekudosten mekaanisia ominai-suuksia. Jäykkyyssäätelyn merkitys korostuu erityisesti tilanteissa, joissa ke-hoon kohdistuu suuria ja/tai äkillisiä ulkoisia voimia, esim. urheiluharjoitte-lussa ja nopeissa korjaavissa liikkeissä horjahduksen tms. seurauksena. Tämän tutkimussarjan tarkoituksena oli selvittää tarkemmin lihas- ja niveljäykkyyden säätelyä erilaisten hyppelysuoritusten aikana sekä lihasväsymykseen liittyvien keskushermostollisten ja perifeeristen adaptaatiomekanismien osuutta rasitta-van hyppelyharjoituksen ja siita palautumisen aikana.

Lihas- ja niveljäykkyyden säätelyä tutkittiin mallintamalla kolmipäisen pohjelihaksen lihas-jännekompleksin ja nilkka- ja polvinivelten toimintaa ja kuormittumista alustaan kohdistuvien reaktiovoimien, videopohjaisen liikeana-lyysin ja ultraäänitekniikan avulla. Hermostollista ohjausta selvitettiin mit-taamalla lihasaktiivisuutta hyppelysuoritusten aikana sekä testaamalla lihasten tahdonalaista aktiivisuutta, voimantuottoa ja refleksitoimintoja ennen ja jälkeen hyppelykuormitusta. Tulokset osoittivat, että nilkkanivelen ja siihen vaikutta-vien lihasten, erityisesti kaksoiskantalihaksen, merkitys on keskeinen koko jalan jäykkyyssäätelyssä hyppelysuorituksissa. Lisäksi havaittiin eroavaisuuksia li-hassolujen ja jännerakenteiden toiminnassa kaksoiskantalihaksen ja leveän kan-talihaksen välillä, mikä edelleen vahvistaa käsitystä yhden ja kahden nivelen yli kulkevien lihasten erilaisista rooleista luonnollisen liikkumisen aikana.

Rasittava hyppelyharjoitus aiheutti välittömän ja myös pitempiaikaisen, useita päiviä kestävän jäykkyyssäätelyn heikkenemisen. Tulokset viittaavat sii-hen, että perifeeristen adaptaatiomekanismien lisäksi myös keskushermostolli-set tekijät saattavat olla osallisena intensiivisen hyppelyharjoituksen aiheutta-maan välittömään lihaksen voimantuoton laskuun ja edelleen jäykkyyssäätelyn heikkenemiseen. Pitempiaikainen voimantuoton ja jäykkyyssäätelyn heikkene-minen puolestaan johtuu todennäköisesti harjoituksen aiheuttamasta lihasvau-riosta ja sitä seuraavasta tulehdusreaktiosta. Yhteenvetona voidaan todeta, että polven ja erityisesti nilkan ojentajalihasten korkea aktiivisuus juuri ennen kon-taktia alustaan (esiaktiivisuus) ja kontaktivaiheen alussa mahdollistaa tehok-kaan jäykkyyssäätelyn hyppelysuorituksissa ja saattaa myös viivästyttää suori-tuskyvyn laskua rasittavan hyppelyharjoituksen aikana.

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76

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