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The Effect of 8 Weeks of Stretching Training on Knee-Joint Range of Motion and Hamstring Muscle Architecture

Preface

This study attempts to investigate the effect of 8-weeks of flexibility training on knee joint range of motion and hamstring muscle architecture during active and passive joint movement. The present study is a part of larger project examining the effects of 24-week stretching.

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Introduction

Stretching training is an important part of many recreational and competitive athletes training and exercise routine. Stretching has been extensively used in order to improve athletic performance (Handel et al., 1997; Worrell et al., 1994, Wilson et al., 1992; Shellock & Prentice, 1985; Smith, 1994), to minimize the risk of injury (Smith, 1994; Worrell et al., 1994; Hartig and Henderson, 1999; Witvrouw et al., 2001; Witvrouw et al., 2003), to rehabilitate injury (Doucette & Goble, 1992) and also to decrease training induced muscle soreness (Buroker & Schwane, 1989, Chen et al., 2001), despite insufficient scientific evidence for such beneficial effects of stretching.

According to the data currently available in the literature, increased range of joint motion has been observed as a consequence of stretching exercise, which is mainly has been attributed to neural adaptations (tolerance to stretch) (Guissard & Duchateau, 2006; Magnusson et al., 1996; Law et al., 2009; Toft et al., 1989; Weir et al., 2005).

Nonetheless, it seems feasible (and has been demonstrated in animal study (Willam & Gold, 1978; Lynn et al., 1998)) that also contractile (muscle) and force bearing tissues (tendons and aponeuroses) may undergo morphological adaptations with sufficient stimulus (Blazevich et al., 2003; Alegre et al., 2005).

Previouslongitudinal studies in humans have applied stretching for limited intervention periods (weeks) and less stimulus (time under stretch) compared to that of animal studies (Ben & Harvey, 2010; Chan et al., 2001; Kubo et al., 2002; Law et al., 2009). However, it is likely that morphological changes to the MTU tissues mayrequirea prolonged stimulus period (Chan et al., 2001). To date, very limited researchhas been carried out to demonstrate the effect of a prolonged period of stretching on muscle architecture.

Aim: The present study, therefore, was designed to investigate the effect of 8 weeks of flexibility training on knee joint range of motion and hamstring muscle architecture during active and passive joint movement.

Hypotheses: It was hypothesized that maximal ROM would increase after 8weeksof stretching exercise. Further it was hypothesized that stretching would elicit increased fascicle length and decreased fiber pennation angle. Finally, it was hypothesized that the above mentioned musclemorphological adaptations would induce a shift of voluntary concentric angle of peak torque towards a more flexed knee joint, while no concurrent changes would be seen in peak torque as a consequence of stretching.

1. Theoretical Background

This literature review will examine the relevant topicsassociated with the effect of various modes of stretching. The topics reviewed in this section cover stretching training, its influences on muscle architecture, factors limiting flexibility, the mechanism governing increases in flexibility (ROM).

Flexibility is known as "the absolute range of movement in a joint or series of joints that is attainable in a momentary effort"(Gummerson 1990). Thus, flexibility is not a general attribute but more specific to a particular joint. Several terms have been used in the literature to describe joint range of motion but most common is simply ROM (Gajdosik, 2001). ROM is defined as the extent by which the constituents of a joint can move relative to each other compared to a reference state of muscle (Gleim & McHugh, 1997; Kisner and Colby, 1996). Reference state can be either "full flexion state" (Gleim & McHugh, 1997) or "neutral position" of the joint. In this text, ROM refers to the angle between neutral position and maximal joint angle in a specific direction.

1.1. Stretching Training

Stretching training has traditionally been considered as an important component of physical fitness (Corbin & Noble, 1980; Taylor et al, 1990). Many athletes use pre-participation routine in order to get prepared for athletic competition. These routines usually consist of a "warm-up" followed by stretching. Briefly, a warm-up is designed to increase the core temperature and to preparethe body for physical activities. There are generally two main types of warm-up: passive and active. The use of heat packs, hydrotherapy, and massage are included in some of the passive warm-up techniques (Wathen, 1987). Therefore, these warm-up techniques are mainly used in sports medicine and physical therapy as preparation for rehabilitation exercises. The active warm-up, however, is used to prepare the athlete for participation in an event. The athlete's muscular power is utilized in the active warm-up to perform light exercises that increase core body temperature without fatiguing the participant. According to Wathen (1987), the warm-up exercise duration should not be too long or high intensity. Stretching is referred to the gradual application of tensile force on a muscle or group of muscles in order to lengthen the muscle and consequently increase the range of motion of a joint. It is often performed as part of a pre-event routine to prepare the body for physical activity. Stretching may be classified into two major group; Static and Dynamic. Both of static and dynamic stretching techniques can be performed in active or passive modes. Passive

technique requires a second person with specific skills. Different stretching techniques have been studied to determine which protocol is most effective with respect to increasing joint ROM (Sady et al., 1982; Smith, 1994).

*Static Stretching.*Static includes lengthening a muscle to the limit of its ROM and holding this position for several seconds.

Dynamic Stretching. Momentum and active muscular contraction are used in dynamic stretching to produce a stretch and it is comprised of movements that are similar to themovements in which the participant will engage (Mann & Jones, 1999).Dynamic stretching gives athletes the ability to being involved, and actively focusing their energy into their pre-event routine. This can be considered as an advantage compared to static stretching, where athletes may find time for conversation, which may hinder the quality of the stretching session. Duration and type of exercises may vary in dynamic stretching, the main focus, however, should be on mimicking activity specific movement patterns (Boyle, 2004).

Even though the effectiveness of static stretching to promote optimal performance has been under debate (Moss, 2002), it should be noted that static stretching is still recommended as a part of a cool down in order to facilitate muscular relaxation, promote the removal of waste products, and reduce muscle soreness (Best, 1995).

The evidence for benefits of stretching, however, is highly limited. It has been suggested in a review study by Shrier (2004) that pre-exercise stretching (acute) decreases force production and velocity of contraction, while the effects of regular stretching (over days to weeks) are opposite.

The effect of stretch duration remains controversial topic. Improved hamstring length in young healthy subjects was reported by Davis et al. (2005) after a program consisting one 30s static stretch three days per week for four weeks. But, longer duration was not tested in this study. Zito et al. (1997) illustratedthose two 15s passive stretching of ankle dorsiflexion was insufficient to producea significant increase in ankle ROM. In another multiple-day study for stretching with duration of 15, 30 and 60 s, Bandy & Iron (1994) reported that 30 and 60s of static stretch of hamstring was more effective than 15s or no stretch (control group).On the other hand, greater rate of gain and a more sustained increase in passive ROM were observed in elderly subjects when one minute stretching of the hamstring muscles was performed compared to 15 and 30 seconds (Feland et al., 2001).Three repeated 45 seconds static

stretches showed no short-term effects on the passive properties of the hamstring muscle-tendon unit (Magnusson et al., 2000). However, five repeated 90 seconds static stretches resulted in a significant change in passive properties on the short term basis (Magnusson et al., 1996b).Therefore,it seems that 60 seconds might have significant effects on flexibility.

According to the data currently available in the literature, it seems that stretching may increase the joint range of motion. This increase appears to be temporary except in the case of intensive stretching exercise. The stretching exercise may also have some side effects, which will be discussed here. The most controversial topics with stretching among researchers are injury avoidance and rehabilitation, muscle soreness decreases/prevention, and impact on muscular strength training and performance improvement.

1.1.1. Stretching in preventing injury

There are no sufficient evidences to endorse stretching to prevent injury among competitive or recreational athletes. Although many authors currently advise that stretching can prevent injury (Ekstrand & Gillquist, 1983; Safran et al., 1989; Garrett, 1990;Herbert & Gabriel, 2002; Mandelbaum et al., 2005; Smith, 1994; Worrell et al., 1994; Hartig & Henderson, 1999; Witvrouw et al., 2001; Witvrouw et al., 2003) and rehabilitate injury (Doucette and Goble, 1992), some have also suggested that stretching does not prevent injury (Shrier, 1999; Pope et al., 2000; Thacker et al., 2004). Some defenders of stretching pretend that the stretching increases the local temperature in the stretched muscles. The rise in the muscular temperature, however, depends only on the development of blood vessels (vascularization) (Gremion, 2005). On the other hand, the stretching leads to increased muscular tension, which may interrupt the blood circulation, therefore the stretching may have reverse effect (Alter, 1996).

A change in the compliance of the muscle-tendon unit is also discussed in connection with stretching. According to Safran (1989), both muscle and tendon determine the ability of a muscle to absorb energy. More energy can be absorbed by a compliant tendonwhen contractile elements are active to a high level, reducing the exposure of musclefibers to trauma. When the compliance of the tendon is low, the forces are transferred to the contractile apparatus. After stretching of the muscle-tendon unit, a lengthening of the tendinous fibers was observed which might lower their effectiveness for shock absorbance (Gremion, 2005). This phenomenon, which is known as creeping effect, seems to be reversible and it is reported to remain more than one hour after stretching (McHugh et al., 1999). Under this condition, the energy is transferred directly to the muscle fibers with an increased risk to generate a muscle

injury and a reduction in flexibility (Gremion, 2005). Based on the aforementioned explanation, it seems that stretching exercise should be recommended in case of warm-up before training (Gremion, 2005).

Even though, flexibility training has been addressed as a mean to reduce muscle soreness, which is known as the sensation of discomfort or pain in the skeletal muscles following physical activity (Alter, 1996; De Vries, 1966; Buroker & Schwane, 1989, Chen et al., 2001), Herbert et al. (2007) have recently noted that stretching before and after exercise does not impart any additional protection from muscle soreness.

1.1.2. Stretching inDecreasing Muscle Soreness

If stretching prevents or reduces muscle soreness, it could then have a positive effect on subsequent physical activity and futureperformance may be relatively unaffected (Andersen, 2005). The effect of pre and post-exercise stretchingon muscle soreness has been reviewed by Anderson (2005). The pre-exercise stretching protocols used in his study consisted of 2 to 10 repetitions held for 20 to 120 seconds for up to 4 days after exercise, whereas, the post-exercise protocols consistedof 4 to 10 repetitions held for 30 to 120 seconds. On average, a reduction of 2% in soreness over the first 72 hours after exercise was observed. This reduced muscle sorenessafter exercise is practically notthat much significant to most patients treated byathletic trainers or other health care professionals (Andersen, 2005). The findings of his review study are in-line with the results obtained withother interventions aimed at decreasing post-exercise musclesoreness (Cheung et al., 2003). In contrast, the results of another review performed by Herbert and Gabriel (2002) do not support the role of stretching in decreasing muscle soreness after exercise.

1.1.3. Stretching and Athletic Performance

In spite of being aware of the issue related to stretching and injuries, flexibility has been recommended in order to improve performance (Beaulieu, 1981; Stamford, 1984; Shellock & Prentice, 1985; Faigenbaum et al., 2005; Yamaguchi and Ishii, 2005). After reviewing 23 studies, including static, PNF and ballistic stretching techniques with both genders, Shrier (2004) revealed that regular stretching may evoke positive long-term performance outcomes. While, stretching prior to performance may elicit insignificant or negative performance outcomes. Shrier's findings were also supported by Haff (2006), Fowles et al., (2000), Kokkonen (1998), and Nelson (2001).

Fowles et al. (2000) reported decreased firing (EMG) and contractile force of the stretched group as results of the prolonged stretching of a muscular groupby conducting a study on the plantar flexor muscle. The decrease in force remained measurable even an hour after the end of the stretching, while the reduction in muscle firing recovered quickly after 15 minutes. Kokkonen (1998) has also tested the effect of stretching in warm-up to the extensor and flexor muscles of the knee. He reported a significant drop in the produced force after passive as well as active stretching, compared to the reference group (without stretching). In a latter study by Nelson (2001) executed on the extensor and the flexor muscles, a decrease of 7 to 8% was reported, respectively. He clearly concluded that stretching before competition event, when an important level of force is required, should be avoided. This effect is called "stretching-induced force deficit" (Cramer et al., 2004a, 2005; Evetovich et al., 2003).

1.2. Influence of Stretching on Muscle Architectures

The architecture of a skeletal muscle is defined as the macroscopic arrangement of the muscle fibers relative to the axis of force exertion (Lieber 1992; Otten 1988; Blazevich & Sharp 2005) affecting conversion of the force and excursion of the muscle fibers into joint actions (Fukunaga et al., 1997). Understanding the muscle architecture is very important when we estimate events that are happening in the muscle from observation of the joint (Fukunaga et al., 1997).

Muscle force production is determined in part by pennation angle (the angle between the muscle fiber and the force generating axis) (Aagaard et al., 2001), whereas muscle excursion and shortening velocity are determined by the length of fiber (Bodine et al., 1982). Thus the architectural features define functional properties of a muscle.It is known that great variation exists in the muscle architecture with respect to fiber length, pennation angle, cross-sectional area, muscle volume within and between individuals. In general, in muscles with short fibers, fibers are packed into the muscles to increase its physiological cross sectional area (PCSA), therefore the muscle can produce more force and use the elastic tendon for energy storage and release providing more efficient muscle tendon movement (Fayad, 2010). Depending on architecture of muscles, smaller volume muscles with short fibers can generate relatively higher force than high volume muscles with long fibers (Fayad, 2010). Fukunaga et al. (1997) has also reported the changes in fascicle length and its impact on high-speed force generation.Pennation angle seems, however, to have little effect on muscle function, particularly when the angles are less than 20˚ (Burkholder, 1994). The reason behind such

characteristic is thatthe force transmitted from the muscle fiber to the tendon and bone (which is a function of PCSA and the cosine of the pennation angle) is becoming only a function of PSCA for the angles smaller than 20˚ (where cosine of pennation angle is getting close to 1). It has also been reported in the literature that when the pennation angle increases, more tissues are attached to a given area of tendon, and slower 'rotation' of the muscle fiber during contraction is possible through a greater displacement of the tendon, thus more force is generated (Aagaard et al. 2001; Kawakami et al. 2001).

The architectural properties of skeletal muscles affect the muscular contraction properties, because fiber, or fascicle, length and pennation angle are strongly associated with differences in the shortening velocity of muscles (Wickiewicz et al., 1984). Therefore, adaptation to different training programs should be adjusted by the changes in muscle architecture specific to each person/athlete (Luis et al., 2006).

Traditionally, the architectural properties of skeletal muscles have been studied using cadaveric tissue, because of difficulties associated with measuring in vivo muscles (Cutts, 1988b; Wickiewicz et al., 1983). More recentlymuscle architecture has been studied in vivo using various muscle-imaging techniques; i.e. magnetic resonance imaging (MR) and ultrasound (US), allowing a direct measurement of the architectural parameters (both fascicle length and fascicle pennation angle) in livingtissue (Rutherford & Jones, 1992; Herbert & Gandevia, 1995; Fukunaga et al. 1997a; Kawakami et al. 1998; Maganaris et al. 1998; Narici, 1999). Although, it has been reported in the literature that studies involved cadaveric tissues are beneficial because the entire muscle can be directly observed and measured (Oxorn et al. 1998), more researchis needed to document the relationship between in vivo and cadaveric skeletal muscle architectural properties. Magnetic resonance imaging has been less successful compared to ultrasonographic studies, when used for in vivo muscle activity (Narici, 1999) that could be because of the restrictions of the instrumentation.The uses of Ultrasonographic scanning have been addressed as the method providing a better understanding of the dynamic nature of skeletal muscle, and could be used to elucidate the biomechanics of muscle contraction (Thomson et al., 2002). To accurately measure the length of short fascicles, which are completely visible in US imaging technique, digitizing software can directly be used. Whereas, for long fascicles it either requires multiple scans along the muscle length to be fitted together (Kawakami et al., 1998), or linear extrapolations have to be performed to estimate the length of the part of the fascicle that cannot be imaged directly due to the limitation of static US imaging (Reeves & Narici, 2003; Blazevich et al., 2009).

Influenceof training on muscle architecture has extensively studied and reported in literatures (Alegre et al., 2006; Aagaard et al., 2001; Blazevich et al., 2003; Blazevich & Giorgi, 2001; Kanehisa et al., 2002; Kawakami et al., 1995; Morse et al., 2008; Rutherford & Jones, 1992; Samukawa et al., 2011). An increase in fascicle pliability and length has been reported after acute stretching (Morse et al., 2008).When the fascicle is lengthened, then the fibers accompanying the fascicle will also be lengthened. According to the length-tension relationship of the sliding filament theory, there should be an ideal length at which muscle fibers contract with greatest force. If the length of a muscle fiber is moved beyond the optimal length, themuscle fiber's sarcomeres will produce less than peak force (Rassier et al., 1999). Therefore, depending upon the muscle's structure and the amount of stretch it experiences, there may be a loss of force due to a change in the resting length (or length before contraction is initiated) within its sarcomeres.

Pennation angle alteration as another possible explanation for force reduction following stretching has also been addressed by Kubo et al., 2001 and Morse et al., 2008. If stretching alters the tendon viscoelastic properties significantly or deformsthe tendon, then the pennation angle of its accompanying musclemay be increased. This increase in pennation angle would be a result of the viscoelastic nature of the tendon (Maruyama et al., 1977). Therefore, when a tendon becomes lengthened, the pennation angle will be increased. It should, however, be noticed that muscle fibers are more affected by stretching and not the tendon. Therefore, decreasedpennation angle is expected as stretching may induce increased laxity of the muscle fiber. When muscle fiber's sarcomeres become more lengthened and induce no changes in tendon viscoelasticity, then the result would be a decrease in pennation angle (Tilp et al., 2011). Previous studies have confirmed a decreased pennation angle during a stretch (Kubo et al., 2001). Morse et al. (2008) have, however, found that pennation angle was increased following a stretching protocol when the muscle was contracted. Thus, the alterations to stretching in non-contracted muscle may differ.Whether pennation angle is increased or decreased with stretching, the lasting effects of stretching on muscle architecture are not fully understood.

In contrast with Morse et al. (2008) and Kubo et al. (2001), in another study performed by Samukawa et al. (2011) pennation angle, and fascicle length were remained unaffected by the dynamic stretching, where increased ankle joint flexibility was explained by lengthening the tendon tissues.

Whether training can change the muscle architectures seems also to be a function of training load and velocity-specific adaptations (Blazevich et al., 2003).Very significant changes have been observed in muscle thickness, pennation angle, and cross sectional area of muscles resulting from heavy resistance training (Alegre et al., 2006; Aagaardet al., 2001; Blazevich & Giorgi, 2001; Kawakami et al., 1995; Narici, 1999), While no-to-slight changes or even decreases in pennation angle have been reported after a period of light resistance training (Blazevich&Giorgi, 2001; Blazevichet al., 2003; Rutherford & Jones, 1992). However, how much heavy does the training loads should be to affect the muscle architectural properties it is still a subject of debates.

Some few and conflicting data exist in literature regarding the duration of resistance training on muscle architectural properties (Alegre et al., 2006; Kawakami et al., 1995; Rutherford & Jones, 1992; Blazevich et al., 2007). Kawakami et al. (1995) using US imaging reported a significant increase in fiber pennation angle from 16.5° to 23.3° in the triceps brachii muscle following 16 weeks of training, while no changes in the pennation angle was observed for quadriceps muscle as a result of 12-week resistance training (Rutherford & Jones, 1992). Recently, Blazevich et al. (2007) have examined the relative contribution of a 10-week concentric and eccentric residence isokinetic knee extensor training on fascicle length of vastuslateralis (VL) and vastusmedialis (VM) on 21 men and women using US imaging. The findings of this study showed that fascicle length of VL adaptation occurred after 5 weeks with no future increase after 10 weeks in both concentric-only and eccentric-only groups. In addition, no significant change in fascicle length of VM was observed. In general, adaptation to the muscle architecture has mainly been reported after long term stretching exercise.

1.3 Mechanism governing increases in flexibility (ROM)

The mechanism governing changes in passive ROM is still debated. Depending upon the duration of the stretching protocol, the proposed theories explaining the changes in joint range of motionarecategorized into two main folds; acute changes that occurs during a short term of flexibility training and last only within a few hours and long term flexibility training which is dealing about more permanent changes which may remain several weeks after flexibility training.

1.3.1 Effect of an acute stretching bout

Studies have shown that there will be an increase in muscle extensibility (increase in muscle length) immediately if they are stretched under a sufficient magnitude, duration or frequency.

It is believed that these changes are due to a viscoelastic deformation, which is limited by muscle's inherent elasticity (Webright et al., 1997; Willy et al., 2001; Weijer et al., 2003; Özkaya and Nordin, 1999).Viscoelastic deformations are produced and measured using various stretching techniques, like static stretches. When a static stretch is applied to a muscle and the muscle is held in the stretched position for a period of time, the ability of the muscle to resist the stretch is gradually declined (Ozkaya and Nordin, 1999; Enoka, 2002; Magnusson, 1998).This decline in resistance is called viscoelastic stress relaxation and it is usually expressed as a percentage of the initial resistance, **Fig. 1.1**. Viscoelastic stress relaxation is equal to the difference of torque between the first time that muscle reaches the final stretch position (peak torque) and the value of the torque at the end of the static stretch holding phase (Magnusson, 1998), **Fig. 1.1**. It is usually calculated using the following equation:

$$\text{Viscoelastic Stress Relaxation} = \frac{\text{Peak Torque - Final Torque}}{\text{Peak Torque}} \qquad \text{..........................} \quad (1\text{-}1)$$
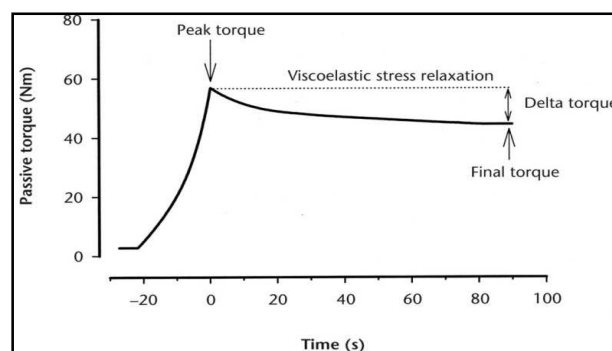

Fig. 1.1. Viscoelastic stress relaxation during static stretch (Magnusson, 1998)

Shrier (2004) suggested that pre-exercise stretching acutely decreases force production and velocity of contraction. In addition to neural adaptations (tolerance), the increase in ROM after acute muscle stretching may be attributed to changes in mechanical properties of the muscle tendon unit (MTU) (Guissard & Duchateau, 2006; Magnusson et al., 1996; Law et al., 2009; Toft et al., 1989; Weir et al., 2005). It is recognized that the MTU extends when forcibly lengthened and recovers its initial length when the force is released. However, due to their viscoelastic properties, these tissues become transiently less stiff after passive stretching (Kato et al., 2010; Safran et al., 1989; Taylor et al., 1990; Magnusson et al., 1996; Halbertsma et al., 1996).Since stiffness is decreased during stretching, less energy is required to move the limb and speed/force of contraction is also increased (Shrier, 2004). In addition to a decrease in tendon stiffness, the reduced MTU stiffness after acute stretching has also been attributed

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to an increase in the extensibility of the muscular portion of the MTU (Morse et al., 2008;Dintiman et al., 1964; Gajdosik, 2001; Liebesman &Cafarelli, 1994).

1.3.2 Effect of chronic stretching training

The biomechanical effects ofchronic stretching programs longer than 8 weeks have not previously been well evaluated. In fact, mostintervention studies on stretching have even been performed within rather short periods of 3 to 8 weeks (Ben & Harvey, 2010; Chan et al., 2001; Kubo et al., 2002; Law et al., 2009), despite that it seems that morphological changes to the MTU tissues mayrequire an even longer intervention period to yield sufficient stimulus (Chan et al., 2001). It remains unclear whether stretching, as it is performed by athletes, can permanently change the material properties of muscles.

Goldspink et al. (1995) and Yang et al. (1997) suggested that improved performance due to long term stretching is linked to stretch-induced hypertrophy. Their animal studies showed that when a muscle is stretched 24 hrs/day, some hypertrophy occurs even though the muscle has not been actively contracting (Goldspink et al., 1995). Muscle length adaptation, and adaptation resulted from a myogenic mechanism (independent of neurological activity) have also been discussed in combination with long term stretching in animal studies:muscles immobilized in shortened positions displayed decreased number of sarcomeres in series (Tabary et al., 1972; Williams & Goldspink, 1978), reduced capacity to resist stretching (Jarvinen et al., 1992), decreased passive resistive forces (Williams & Goldspink, 1978), decreased extensibility (Tabary et al., 1972; Williams & Goldspink, 1978) and increased passive elastic stiffness (Tabary et al., 1972; Williams & Goldspink, 1978). Increased amount of sarcomeres in series has been addressed in the above mentioned studies as increasing muscle length. It is also clearly illustrated by Gadosik (2001) that increased the healthy muscle´s functional length, improved its ability to withstand a passive load and its passive elastic stiffness is expected as effects of long term stretching, without changing the viscoelastic stress relaxation properties. In a similar study, increased passive stiffness following 6–week flexibility training was reported by Reid and McNair (2004). In their work, increased passive resistive forces at end range are observed along with increase in the passive knee extension ROM. These results support the data found by Halbertsma and Goeken (1994) and Magnusson et al. (1996c). However, increased passive elastic stiffness reported in Reid and McNair (2004) was not observed in the study conducted by Magnusson et al. (1996c), where no change in the force-length curve was found. The difference observed between Reid and McNair (2004) and Magnusson et al. (1996c) was discussed in connection to the

differences in the age of the subjects. In another study performed by Gajdosik et al. (2007) on long term calf flexibility training (6-week training), passive elastic force adaptation and increased absorbed passive elastic energy were reported, while no significant increases in passive elastic stiffness was observed. They believed that passive adaptation could be due to remodeling of connective tissue or by adaptation in the non-contractile proteins of the sarcomeric cytoskeleton. Increased joint angle as a result of long term flexibility training of the hamstring muscles has also reported by Magnusson et al. (1996c), even though, no changes in stiffness of the linear portion of the torque-angle curve, energy or passive torque at a predominated joint angle was observed. In latter study, after 4 weeks of flexibility training for the hamstring muscles, increased passive hamstring ROM and increased passive moment was addressed, but without any changes in elasticity (no shift of passive torque-angle was found)(Halbertsma and Goeken, 1994). The generally suggested mechanism for increases passive ROM was an altered stretch tolerance rather than any change in mechanical properties (Magnusson et al., 1996c; Halbertsma & Goeken, 1994). It has also been supported repeatedly in several studies involving hamstring muscles, where various stretching and testing methods were utilized (Chan et al., 2001; Folpp et al., 2006; Gajdosik 1991; Reid & McNair 2004; Ben and Harvey 2009; Law et al., 2009).

According to Hutton (1992), the passive properties of connective tissues and tendons are changed after long term stretching (20 days). Magnusson (1998), however, believes that these changes in material properties is observed only when a decrease in resistance can be demonstrated at the same joint angle or a greater joint angle can be achieved with the same resistance.

Increase in static ROM associated with a decrease in passive tension was experimentally shown by Toft et al (1989), who found a 36% decrease in passive tension of the plantar flexors after three weeks of regular calf stretches. The relationship between static ROM and passive tension has been further supported by McHugh et al (1998). These researchers demonstrated that maximum static hip flexion ROM was inversely correlated with the passive tension of the hamstrings during the mid-range of hip flexion. This suggests that the ease with which the muscle can be stretched through the mid-ROM is increased if the maximum static ROM is improved. The concept that increased static ROM results in more pliant mechanical elastic properties of the muscle, suggests that static stretching is beneficial to sports

performance. The literature often mentions changes in the passive properties of connective tissue and tendon as the mechanism for long-term improvement in flexibility (Hutton 1992).

Plastic deformation of connective tissue is another proposed theory forincreased ROM which has been subjected to stretching is due to "plastic" (Chan & Hung, 2001; Feland et al, 2001) or "permanent" (Chan & Hung, 2001; Draper at al., 2004) deformation of connective tissue. Based on this theory, if the connective tissues of a muscle are stretched with intensity beyond their elastic limit (into the plastic region) of the torque/angle curve, once the stretching force is removed, the muscle would not return to its original length and retains it lengthened state. The plastic deformation is illustrated on the passive length/tension curve by a decrease in the slope above the yield point. (Enoka, 2002), **Fig. 1.2**. It seems that no plastic deformation occursduringnormal stretching (Weppler & Magnusson, 2010).
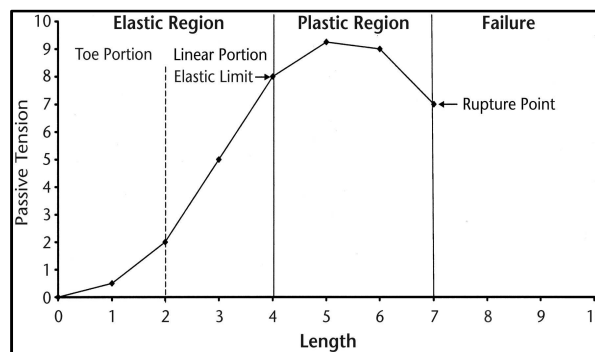

Fig. 1.2. Model passive length/tension curve for Biological tissue (Weppler & Magnusson, 2010).

Animal studies have also shown that muscles are able to adapt to new functional lengths by changing the number and the length of sarcomeres. By such changes muscles can optimize their force production at the new functional length. These studies have demonstrated that when muscles are kept in a fully extended position, the number of sarcomeres in series is increased. At the same time, a decrease in sarcomere length is documented (Williams & Goldspink, 1978). The overall result of increase in the number of sarcomers and decrease in the sarcomer length is believed to be no change in muscle length.In the same way, when a muscle is immobilized in shortened positions, the number of sarcomeres in series decreases and a decrease in muscle length is documented, but the muscle length returns to its original level decrease after recovery from immobilization (Tabary et al, 1972).

Previous work suggests that contraction of muscles due to a neuromuscular "stretch reflex" is able to limit muscle extension during static stretching procedures (Spernoga et al, 2001). It is

believed that if a static stretch is slowly applied on a muscle, it would stimulate neuromuscular reflexes and that induces relaxation of muscles which are subject to static stretch. This process leads to an increase in the muscle extensibility. It has also been suggested that neuromuscular reflexes adapt to repeated stretch over time, which enhances the ability of the stretched muscle to relax. Fast and short stretches of muscles that are in mid-range position activate stretch reflexes, producing a muscle contraction of short duration (Chalmers 2004). Magnusson et al. (1996) evaluated the effects of a single "contract-relax" stretch and in stretching studies lasting for only 3 and 6 weeks'. He did not observe any shift of passive torque/angle curves. Therefore, the observed increase in end-range joint angles could not be due to neuromuscular relaxation.

Most of the mentioned theories have dealt with a mechanical increase in length of the stretched muscle. However, recently proposed sensory theory suggests that increases in muscle extensibility are because of a modification of sensation only. Increases in muscle length during stretch application because of viscoelastic properties of muscle have been reported in studies,evaluating the biomechanical effect of stretching. However, some believe that this length increase is transient and its magnitude and duration dependent upon the duration and type of stretching applied on the muscle (Magnusson et al, 1996, Halbertsma et al, 1996). In these kinds of studies, it has been suggested that increases in muscle extensibility immediatelyafter a single stretching session and after short-term (3- to 8-week) stretching programs are due to an alteration of sensationand not to an increase in muscle length. However, the biomechanical effects of long-term (≥8 weeks) and chronic stretching programs have not yet been evaluated (Halbertsma et al, 1996; Nelson & bandy, 2004)

1.4 Factors Limiting Flexibility

An athlete's flexibility or ROM is restricted by a number of anatomical and physiological factors. Moreover, some external factors may also influence the flexibility; i.e. the temperature, the time of day, age, gender, clothing and equipment (Gummerson, 1990). In this section, the most common factors limiting one's flexibility are discussed, which muscle mass, excess fatty tissue, and connective tissue (physical injury or disability).

1.4.1   Joint Structure

The degree of range of motion at the joint is mainly affected by joint structure. Some have a greater range of motion compared to the others; i.e. the ball and socket joint of the shoulder has the greatest range of motion of all the joints (Anthony & Kolthoff, 1975).

1.4.2   Age & Gender

Age has a great impact on flexibility levels; flexibility decreases with age. Because when a person getsolder, skeletal muscles become less elastic. Aging skeletal muscles undergo the process of fibrosis, under which they develop increasing amounts of fibrous connective tissue. Fibrosis makes the muscle less flexible, and the collagen fibers can restrict movement (Anthony & Kolthoff, 1975). Increased level of calcium deposits and cross-links in the tissues, increase in the level of fragmentation and dehydration, changes in the chemical structure of the tissues and the replacement of muscle fibers with more fatty fibers are all suggested as different factors causing decreased flexibility as age increases (Alter, 1996). Females tend to be more flexible than males (Gummerson, 1990).

1.4.3   Connective Tissue

The connective tissues surrounding muscles are limiting factors affecting ROM. Magnusson (1998) has illustrated that the intramuscular connective tissue, which are responsible for force transferring, distribute stress and prevent overstretching. Animal studies conducted by Johns and Wright (1962) on the joint resistance during passive motion showed 47% change in joint capsule, 41% in the fascia surrounding muscle, 10% in tendon and 2% in skin. According to Alter (1996), efforts to increase flexibility should be directed at the muscles fascia, because it has the most elastic tissues and because ligaments and tendons are not intended to stretched very much at all (since they have less elastic tissue). When a muscle or joint remain inactive, chemical changes in connective tissue may take place which may restrict flexibility. Also, overused connective tissue may become fatigued and may also tear. This can also limit flexibility. Unused or underused connective tissues, however, provide significant resistance and limit flexibility (Alter, 1996).

1.4.4   Proprioceptors

The capacity of the neuromuscular system to inhibit the antagonists (those muscles being stretched) may also influence flexibility (Dick, 1997). Two important proprioceptors have been considered involving in the mechanics of stretching and flexibility; muscle spindles and golgi tendon organs (GTO). The muscle spindles are located within the muscle fibers and monitor changes in muscle length. The muscle spindles activate the stretch reflex, which is the body involuntary response to an external stimulus that stretches the muscle (according to National Strength and Conditioning Association) and causes a reflexive increase in muscular activity. The muscle spindles and the stretch-reflex response both have been referred to as restricting motion. The GTOs are located near to the musculotendinous junctions and are

sensitive to increase in muscle tension. When the GTO is stimulated it causes a reflexive relaxation in the muscle. When this relaxation occurs in the same muscle that is being stretched, it is referred to as autogenic inhibition and can facilitate the stretch (Leighton, 1964). Autogenic inhibition can be induced by contracting a muscle immediately before it is passively stretched a technique used in PNF stretching.

1.4.5 Previous Injuries

Injuries to muscles and connective tissue can lead to a thickening or fibrosing on the affected area. Fibrous tissues seem to be less elastic and can lead to reduced ROM (Gummerson, 1990).

1.4.6 Temperature

The temperature of the joint and its connected tissues are influencing the flexibility, which can be discussed in combination to warm-up effects. Joints and muscles offer better flexibility at body temperatures that are 1 to 2 degrees higher than normal (Gummerson, 1990); A warmer temperature of the place where one is training is more conducive to increased flexibility.

To sum up, the literature demonstrates increased passive ROM as a result of stretching exercise, although the results and proposed mechanisms are conflicting. In order to provide a proper recommendation for athletes regarding the flexibility program and its effect on the performance, more research is needed in this area.

2   Materials & Methods

2.1.   Design

The present study was a randomized control trial (within-subject control,stretch versus non-stretch leg), investigating the effects of 8 weeks of hamstring stretching training on joint range of motion (ROM), muscle architecture, and contractile properties. All the assessments, described in section 2.3, were performed before and after the stretching intervention.

2.2. Subjects

12 subjects volunteered for the study, but seven participants withdrew for different reasons mostly pertaining to practical matters. Therefore, the intervention was completed with five healthy subjects (3 male, 2 female). Inclusion criterion was 17 yrs< age <35 yrs. Participants were not accustomed to regular stretching exercise in the preceding 5 yrs. The main exclusion criterion was if subjects had performed previous stretching exercise on at least a weekly basis, maximum 3 times a week for 10 min. Moreover, the participants were excluded with:

- Hamstring flexibility$\geq$-5 (Sit-and-reach test described below)
- A history of injury to hamstring muscle tendon units over the last 6 months
- Any orthopedic disorders

Table 2.1 shows the mean values of body mass, height, standing leg length from trochanter major to floor, and lower leg length from the lateral epicondyle of the femur to the center of the lateral malleolus, measured for the subjects.

Table 2.1. Anthropometric measurements

	Mean±(SD)
Age	25.6±5.5
Height	175.5±9.1
Bodymass	73.5±11.3
standing leg length	89.0±6.9
Lateral epicondyle	47.8±14.6

2.2.1.   Ethics

Prior to the study the subjects received a written consent form describing the procedures as well as the purpose of the study. Each subject agreed and signed the consent form whichwas approved by the regional committee for medical and health research ethics. After the screening procedure (sit and reach test) and before inviting the subjects for the initial test, they were also informed about the risks associated with this study. Participation in the study

was voluntary and subjects could withdraw at any time without providing a reason. All experimental procedures were performed in accordance with the declaration of Helsinki.

2.3. Methods

All subjects were instructed to reach the lab without significant physical activities (running, biking, etc.). Before starting the tests, they were asked to sit for 10 min to minimize any potential effects of walking to the lab. For each experimental session (pre & post), the subject stayed about three hours at the lab. Prior to the first experimental session, each subject underwent familiarization. Each participants received a written informed consent form (Appendix 1) describing the goal of the study, and risk associated with the study. The following measurements were performed for all subjects before and after the 8 week stretching intervention.

2.3.1. Hip joint ROM assessment (hamstring flexibility)

General hip joint ROM (hamstring flexibility) was assessed by use of the sit-and-reach test (Canadian Society for Exercise Physiology, 2003): The subject sat on the floor with hips flexed, knee joints fully extended, and foot-soles in full contact with the box (ankle at anatomically neutral position, feet: shoulder-width apart). The subject reached symmetrically forward with extended arms towards and/or beyond the feet. The reach was kept for 2s while reaching distance was recorded as the distance between the most distal finger tipand the plane of the foot-sole.

2.3.2. Ultrasonographic recording

Potential muscle architectural changes of the biceps femoris muscle were investigated by ultrasonography. Longitudinal images were obtained using ultrasound (HD11XE, Philips, USA) with a linear-array probe (L12-5;32HZ wave frequency with 30 mm scanning length). The width and depth of resolution of the images was 4.43 pixel/mm. The subjects were placed in a prone position with knees extended.The probe was placed directly on the skin above the middle aspect of the muscle (i.e. 50% along the thigh length). A transmission gel (aqua sonic, Parker laboratories, USA), was used as a coupling agent to provide acoustic contact.The probe was carefully adjusted such that a clear image of the pinnate fascicle structure was obtained and further such that the profound aponeurosisof insertion was clearly visible. Two images were recorded, Images were analyzed with publicly available imaging software (image j 1.45s) National Institutes of Health; free to download from

(http://rsb.info.nih.gov/ij/download.html) to measure the fascicle length and pennation angle for biceps femoris(Chleboun, et al., 2001).

From each ultrasound image, the fascicle length (FL) and pennation angle (PA) was measured using image analysissoftware (Image J 1.42). FL was measured from a clearly visible fascicle bundle lying between the superficial and deep aponeuroses (Fukunaga et al., 2001; Kawakami et al., 2002). The end of fascicle line was extended off the acquired ultrasound image, and FL was measured as FL=FLmeasured+FLestiomated (**Fig. 2.1**). PA(μ) was also measured as the angle between the fascicle and its insertion on the deep aponeuroses (**Fig. 2.1**) (Abellaneda et al., 1999; Behm et al., 2001).
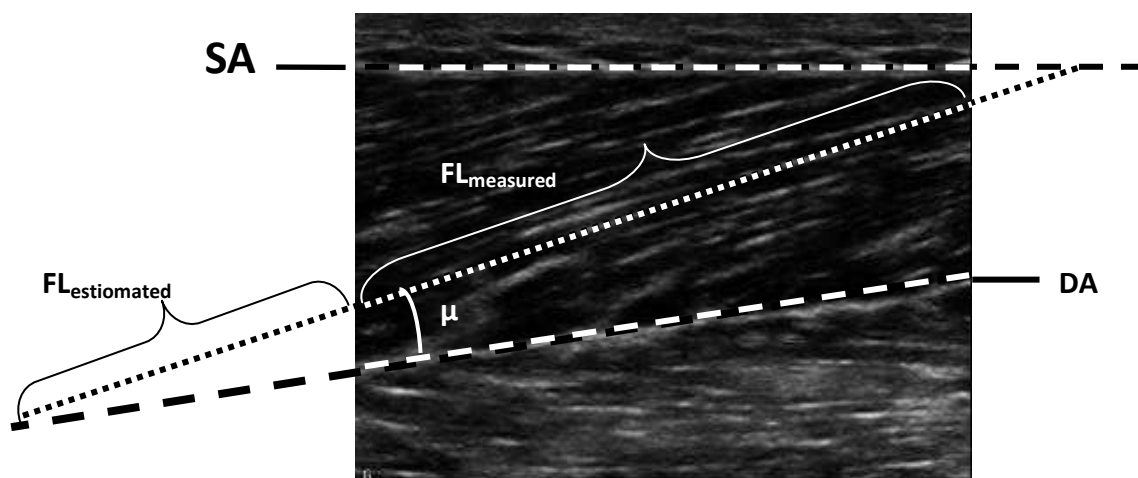

Fig. 2.1. Typical example of Ultrasound image of biceps femoris. Showing superficial aponeurosis (SA), deep aponeurosis (DA), fascicle (fas). FL and PA (μ) were also shown.

For long fascicles linear extrapolations was performed to estimate the length of the part of the fascicle that cannot be imaged directly due to the limitation of static US imaging (Reeves &Narici, 2003; Blazevich et al., 2009).

2.3.3. Isokinetic dynamometry

Anisokinetic dynamometer (Techno gym Rev 9000, Cesena, Italy) was used to measure knee flexor and extensor muscle contractile properties.Subjects were seated on a sloped surface to increase hip joint angle (fixed at appr. 100° for all the subjects) (**Fig. 2.2**) with arms crossed over the chest during all testing. The seat slope angle of 25° was equal for all subjects. Straps for stabilization were placed over the waist, chest and distal thigh. The rotational axis of the dynamometer was aligned with the axis of the knee joint. To make sure that the seated position of the subjects was the same in pre and post test, after fixing the leg and aligning the

dynamometer in pre-test, its position was recorded and fixed for the post test. Gravity calibration for limb mass was also performed before each isokinetic assessment in accordance with the manufactures instruction.

The lower leg was fixed to the lever arm of the dynamometer by a cuff. The cuff was placed around the ankle, and the distance from the center of rotation to the center of the cuff was registered.

Knee extension range of motion was defined for each participant individually bypassively moving the dynamometer while subjects kept muscles relaxed.For safety of the subjects, mechanical stops were placed at the beginning and end of the full active range of motion

Fig. 2.2. *Position of subject on an isokinetic dynamometer (Techno gym Rev 9000)*

Subsequently all subjects were tested in the following isokinetic tests:
- Passive torque at maximally tolerated knee joint extension
- Passive torque to a predetermined knee joint angle
- Voluntary concentric knee extension and flexion

Passive torque at maximally tolerated knee joint extension

The dynamometer was set in passive mode for knee flexion and extension. The subjects were asked to relax their muscles and to keep their eyes closed,while the knee joint was extended, starting from 100° of knee joint flexion.The subjects were notified of the coming procedure, and to signal onset of pain.The knee joint was moved passively with increments of 1-2° per

sec. until onset of pain. The corresponding knee joint angle was noted asthe maximum tolerated knee extension angle (hereafter denoted "KA_{max}").Hereafter, the leg was moved passively to KA_{max}, at a speed of 5˚/sec while resistance force was sampled. This experiment was performed twice.Concurrently, the stretch tolerance of the subjects was quantified usinga visual analog pain scale (VAS). The VAS is a 100 mm horizontal line representing no pain at left end and worst imaginable pain at right end of the line. Subjects were asked to mark a point through the horizontal line that best represented the pain experienced during the maximal and predefined passive tests. The VAS score was collected immediately after passive tests.

Passive torque to pre-determined knee joint angle

A predetermined joint angle (KA_{max}-10°) was identified individually for each subject, and for each leg. Thesubject was positioned in a similar manner as described above, and the leg was moved passively (knee extension) to the predetermined angle and back to neutral position at a speed of 5°/sec while passive resistance force was registered. The experiment was repeated twice, and VAS score was assessed.

Voluntary concentric knee extension and flexion

Active knee extension and flexion tests were carried out in the same joint range of motion as the passive tests for each subject and leg. (KA_{max}-10°):Three sub-maximal warm-up trials proceeded four maximal muscle actions at constant velocity, with a speed of 5°/sec, (concentric knee extension and flexion) and a 5 sec.a period of rest was allowed between the tests. The effort with the greatest amount of peak torquewas selected for further analysis. The outcome parameters for analysis were pick-torque (Nm), the joint angle at peak-torque (°), leg extension range of motion (°).

For isokinetic active extension and flexion tests, twosimilar torque curveswith highest single peak values werepicked for further analysis. Then, similar to passive tests, the mean of maximal peak torques in two test trials was used for data analysis.

2.4. Stretching protocol

After the pre-test session, all subjectsunderwent 8 weeks of daily hamstring stretching exercise; four sets of60 sec static stretching (Handelet al., 1997). In an initial familiarization session, subjects were instructed on how to perform the stretching exercise.

2.4.1. Warm-up exercise

Prior to stretching, 3-4 min of warm-up was carried out. Warm-up exercises as well as their descriptions are as follow:

1. Jumping jack; this exercise was performed by jumping to a position with the legs spread wide and then returning to a position with the feet together, **Fig. 2.3(a)**. The exercise was repeated 30 times.

2. Diagonal Lunge; hands were placed on waist. From a standing position, subjects were taken a step forward and diagonally to non-training leg. The training leg was extended enough to stretch the hamstringing muscles during the forward walking lunge exercise. The front leg bent and heels were kept fully in touch with the ground. The trunk bent forward to place hands on the ground. After pausing for a while in that stretching position returned to the starting position, **Fig. 2.3(b)**.This exercise was also repeated 8times. Lateral lunge: This exercise started by standing balanced with arms placed on waist. Non-training leg moved forward and crossed the other leg. Knees bent slightly and arms stretched to touch the ground. After pausing for a while in that stretching position returned to the original position, **Fig. 2.3(c)**.This exercise was also repeated 8times.
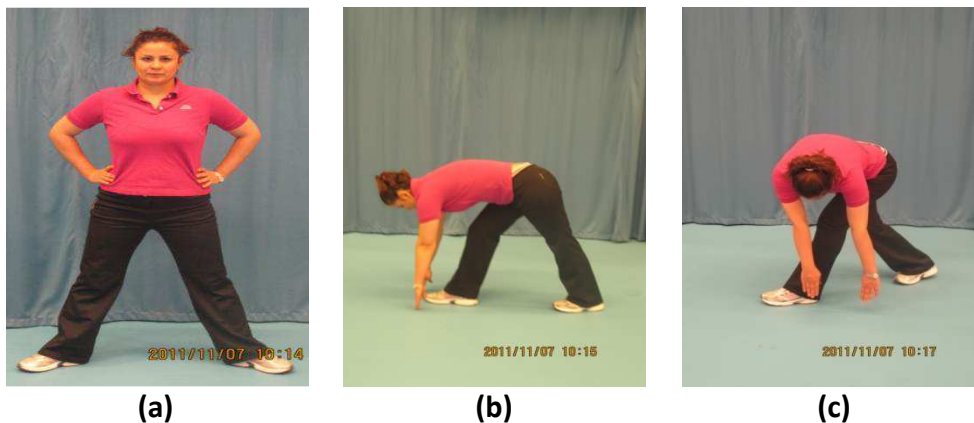

(a)   (b)   (c)

Fig. 2.3. Position of subject in warm-up exercises; a. Jumping jack, b. diagonal lunge, and c. lateral lunge

2.4.2. Static stretching

Subjects were asked to place the heel of the leg on the step to stretch the hamstring muscles. Hands placed on the hips, head held in the natural position, and the stretched leg kept fully extended. Subjects were also asked to move their trunks forward at the pelvis until they felt a hamstring sensation without pain, **Fig. 2.4**. After pausing for 60 sec. in that stretching position, they returned to the starting position. Stretching exercise was repeated 4 times. Each

repetitions separated by 60sec. Subjects performed daily stretching trainings for totally 8 weeks. Subjects kept a standardized training diary to monitor their training. Moreover, subjects were contacted twice a week by an investigator to ensure compliance.All participants were instructed to refrain from exercise for 24hrs prior to post-test. Pre and posttests were conducted by the same investigator and at the same time of day for individual subjects.When performing the post test, the participants were blinded from the prior test results.

Fig. 2.4. Illustration of the hamstring stretching exercise

2.5. Statistical Analysis

ROM was analyzed before and after stretching using the Student´s paired t-test. Changes in active and passive torque, FL, and PA before and after the stretching exercise were analyzed by a one-way analysis of variance (ANOVA). For all the ANOVA analysis, a Torkey´s post hoc test was used to identify the changes among the means. For all analyses, the level of statistical significance was set at $p < 0.05$. Data are reported as mean $\pm$ SD (standard deviation) in the text and displayed as mean $\pm$ SE (standard error) in the figures.

SPSS (Statistical Package for the Social Science, version 18) software was used to analyze the results. The mean peak torque, standard deviation (SD) of torque, and the coefficient of variation (CV) of torque (SD/mean×100) were calculated from each variable of interest.

3. Results

3.1. Hip joint ROM assessment

Hip joint ROM measured after 8-weeks stretching was compared to the pre-assessment before stretching program, **Fig. 3.1**. ROM was found to be statistically different ($p < 0.05$) between pre-and post-tests. Before training, the mean value obtained from the sit and reach test, was -0.2 ± 8.6 cm, while it increased to 8.4 ± 7.7 cm after 8 weeks of stretching exercise.
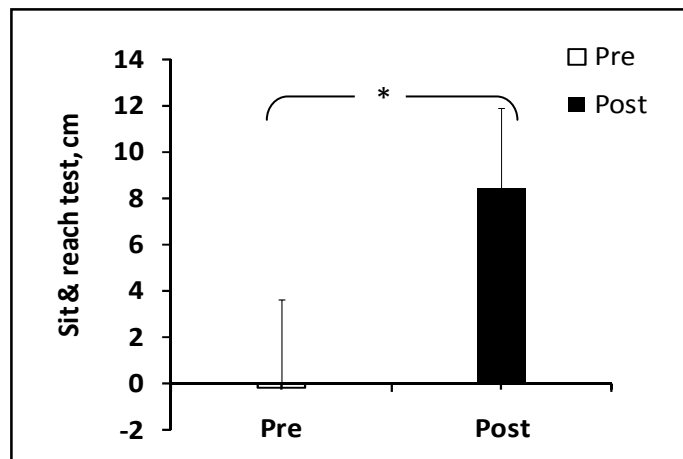

Fig. 3.1. *Mean sit and reach test (ROM) for pre- and post-tests. Values are mean±SE*
* Significant difference between pre- and post-stretching values: *P<0.05

3.2. Ultrasonographic recording

Data collected by use of ultrasonographybefore and after 8 weeks stretching training are presented in **Figs. 3.2**, and **3.3** for both control and training legs.**Fig. 3.2** illustrates that the fascicle length remained unchanged.
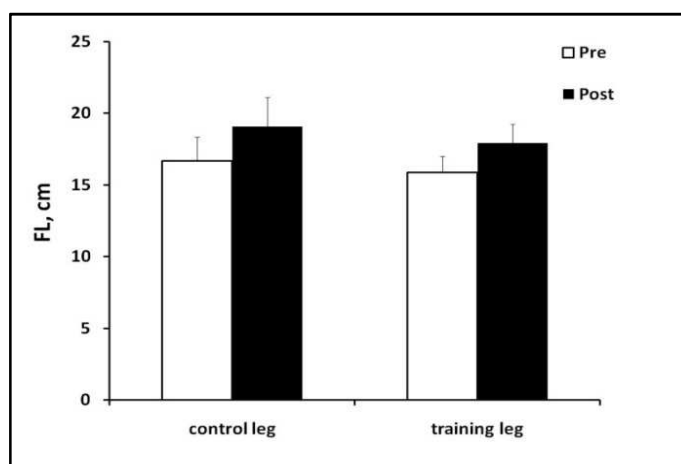

Fig. 3.2. *Mean FL for pre- and post-stretching for both control and training legs.Values are mean±SE*

When stretching exercise was completed, the changes in pennation angle did not show any significant differences both between and among groups (**Fig. 3.3**).
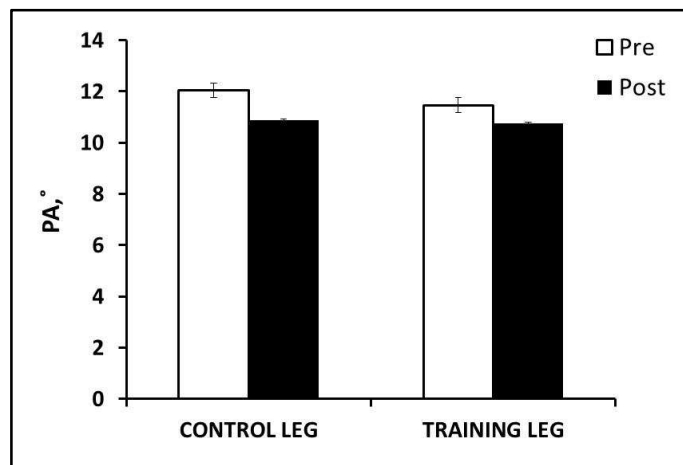

Fig. 3.3. *Mean PA(°)for pre- and post-stretching for both control and training legs.Values are mean±SE*

3.3. Isokinetic test

The influence of the stretching program upon the muscle strength and joint angle ROM are presented in **Figs. 3.4** to **Fig. 3.13.** The averaged mean values (± standard error of the mean) for the isokinetic measurements are also shown in the figures.

3.3.1. Passive torque at maximal tolerated and at predetermined joint angle of knee

In general, the statistical analysis for both torque and angle of peak torque at maximal tolerated joint angle of the knee indicated no significant differences between pre- and post-stretching ($P>0.05$), **Figs. 3.4** & **3.6**. After the stretching period, four subjects seemed to display lower peak torque at maximal tolerated joint angle of knee in post-test compared to pretest assessment, as shown for one subject in **Fig. 3.7**.
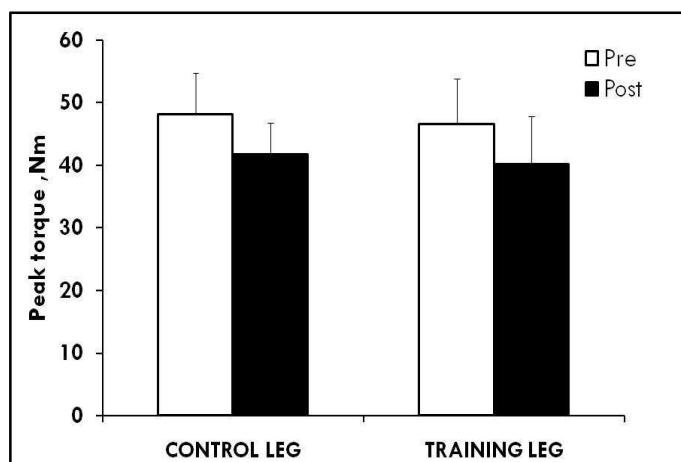

Fig. 3.4. *Passive peak torque at maximal tolerated joint angle of knee for pre- and post-tests in both control and training legs.Values are mean±SE*
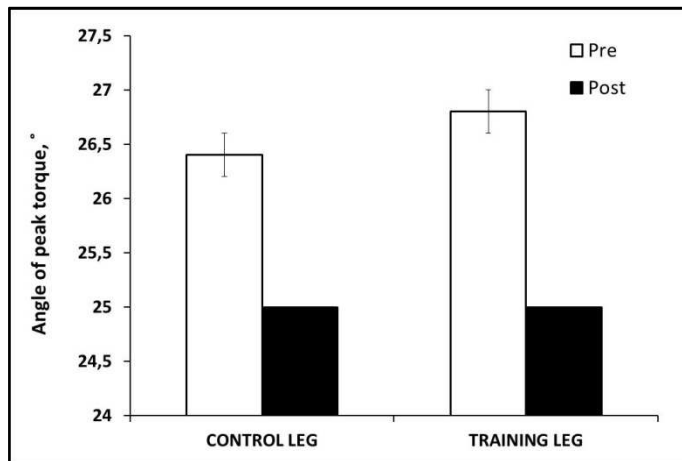
Fig. 3.5. *Angle of peak torque at maximal tolerated joint angle of knee for pre- and post-tests in both control and training legs.Values are mean±SE*
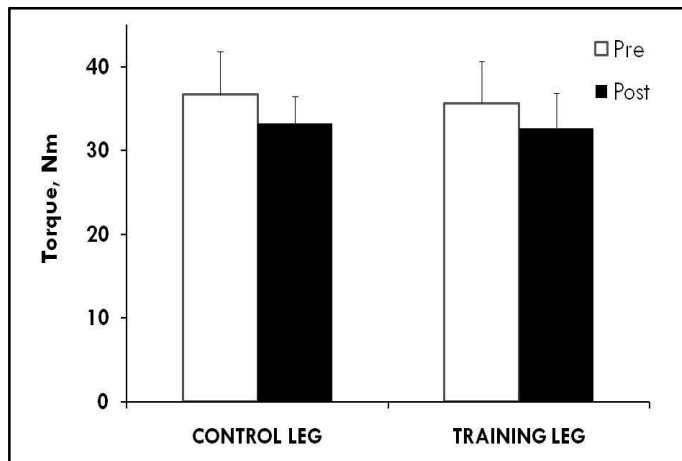

Fig. 3.6.*Torque at a predefined joint angle for pre- and post-tests in both control and training legs.Values are mean±SE*
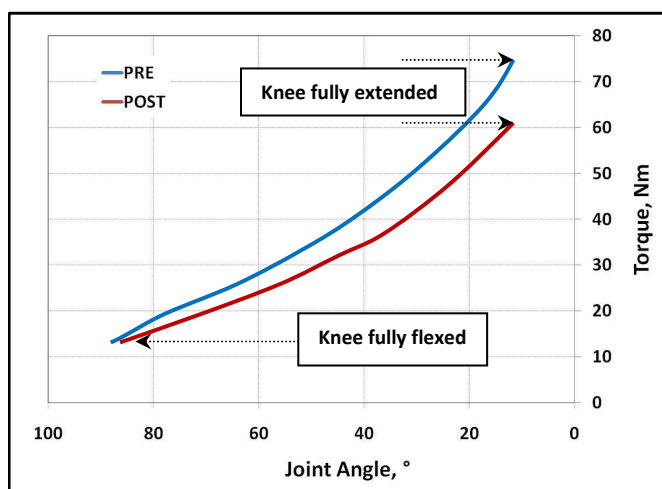

Fig. 3.7. *An example of passive torque as the knee joint is extended passively for pre- and post-tests in training leg of one subject*

Pain perception measured by VAS scale during the passive test showed no significant changes in passive tension after 8-week stretching; confirming that all subjects experienced the same level of pain during the post-test at a given range of motion compared to the pre assessments, **Fig. 3.8**.
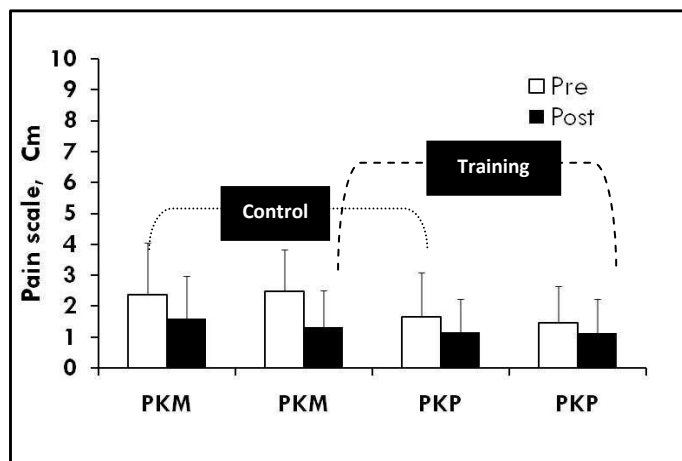
Fig. 3.8. Mean VAS score from isokinetic passive tests (maximal and pre-defined joint angle) for both control and training groups. Values are mean±SE. PKM: Maximal Passive Knee, and PKP: Pre-determined Passive Knee.

3.3.2. Voluntary concentric knee extension and flexion

The statistical analysis for maximum torque measured at knee extension indicated no significant differences between and within groups (P>0.05), **Fig. 3.9**.

Fig. 3.9. Mean peak torque at voluntary concentric knee extension measured from isokinetic test for pre- and post-tests in both control and training legs.Values are mean±SE

ANOVA analysis also indicated no significant changes in mean angle of peak torque at voluntary concentric knee extension measured from isokinetic testwithin and between groups, **Fig. 3.10**.

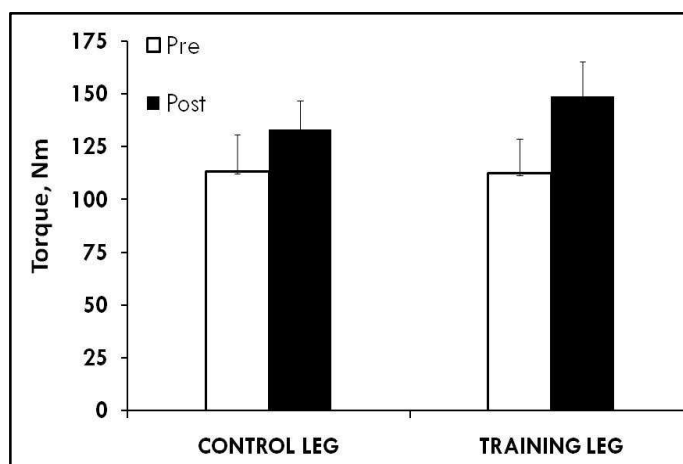***Fig. 3.10.*** *Mean angle of peak torque at voluntary concentric knee extension measured from isokinetic test for pre- and post-tests for both control and training legs.Values are mean±SE*

In addition, the results of active dynamometer tests showed that stretching exercisedid not cause significant changes ofmaximum force development in flexion in both control and intervention legs, **Fig. 3.11.** The angle of peak torque, however, increasedsignificantly in the intervention leg, not in control leg; increased from 61.6±2.0 (SD) in pretest to 68.5±6.3(SD) in posttest, **Fig. 3.12**.
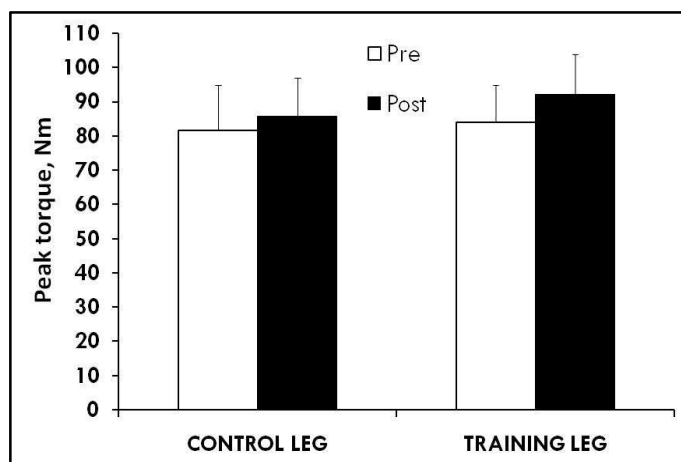
Fig. 3.11. *Mean peak torque at voluntary concentric knee flexion measured from isokinetic test for pre- and post-tests for both control and training legs.Values are mean±SE*
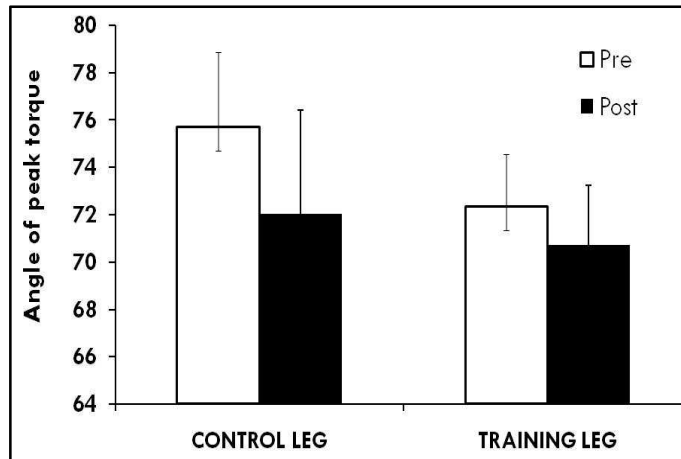
31
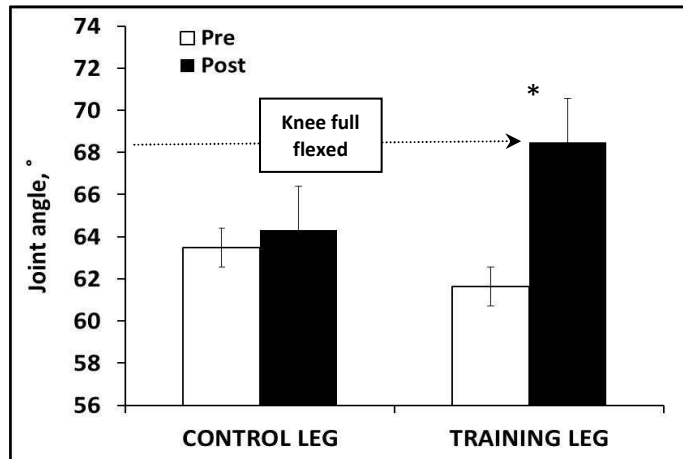
Fig. 3.12.*Mean Angle of peak torque at voluntary concentric knee flexion measured from isokinetic test for pre- and post-tests for both control and training legs.Values are mean±SE*

A significant shift of voluntary concentric angle of peak torque towards a more flexed knee joint is also shown in **Fig. 3.13** for one subject as an example**.**
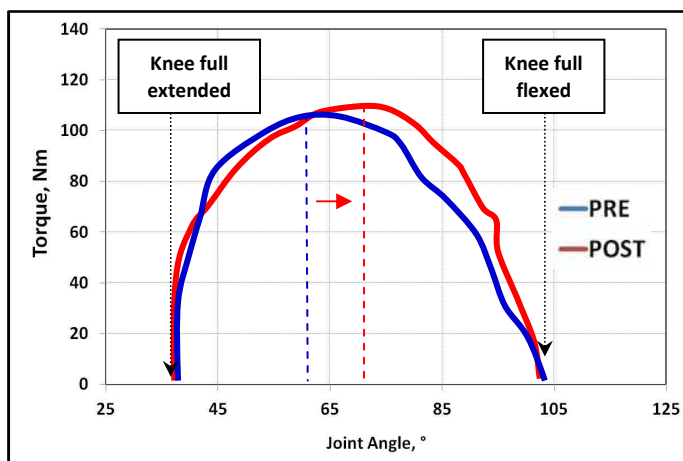
Fig. 3.13.*Voluntary concentric angle of peak torque during knee flexion. Training seems to shift the curve to the right such that angle of peak torque occurs at a more flexed knee joint (data from one subject).*

4. Discussion

Main Findings

The participants in this studyincreased their maximal knee joint range of motion (ROM) after 8 weeks of training. The increased ROM was accompanied by a significant shift of angle of peak torque towards a more flexed position (increase in angle of peak torque, i.e peak torque occurred at a more flexed knee joint position)in the intervention leg during active concentric contraction.

Maximal Knee Joint Range of Motion (ROM)

Several studies (Guissard & Duchateau, 2006; Magnusson et al., 1996; Law et al., 2009; Toft et al., 1989) have reported increases in joint range of motion following a bout of stretching. The results of this study support the previous findings. 'Sit-and-reach' test measurement was conducted to indirectlyinvestigate the changes in ROM. A significant increase in test person long after 8-week flexibility training is an indication of increased ROM.

Four main mechanisms have been postulated to explain the stretching-induced increases in joint range of motion: (a)Viscoelastic deformation (Magnusson, 1998), (b) neural adaptation (tolerance) such as motor unit activation (Guissard & Duchateau, 2006; Magnusson et al., 1996; Law et al., 2009; Toft et al., 1989; Weir et al., 2005), (c) mechanical factors such as decreases in muscle stiffness that may affect muscle's length-tension relationship and/or sarcomeres shortening velocity (Cornwell et al., 2002; Cramer et al., 2004a, 2005; Evetovich et al., 2003; Fowles et al., 2000; Kokkonen et al., 1998; Nelson et al., 2001a, 2001b; Nelson &Kokkonen, 2001), (d) changes in muscle's contractile properties such as increases in fascicle length and decreases in pennation angle.

To quantify the hypotheses of this study, the changes in joint angle of peak torque as well as pain and tolerance were evaluated.

Changes in Mechanical Properties of MTU

According to Fowles et al. (2000), altered mechanical properties of the MTU may affect the muscular force generating capacity rather than the neural factors. He and his coworkershypothesized that stretching could have changed the length-tension relationship and/or the plastic deformation of connective tissues. Cramer et al. 2004 has also hypothesized that stretching-induced changes in thelength/tension relationship may be manifested throughchanges in the torque versus range of motion relationship, which, in turn, may affect

the joint angle at PT. Therefore, to check the hypothesis of the present study, the shape of angle-torque curve was investigated in pre- to post-stretching. Our results indicated that despite the stretching induced significant increases in joint angle of peak torque in intervention leg, there were no changes in the peak torque as a result of stretching. A significant increase in angle of peak torque can be explained by morphological adaptation of force bearing tissues (tendons), although the pennasion angle remained constant after stretching. Maruyama et al. (1997) illustrated that when a tendon becomes lengthened (altered the tendon viscoelastic properties), the pennation angle will be increased. However, similarly toSamukawa et al. (2011), where both pennation angle and fascicle length were remained unaffected by the dynamic stretching, increased joint flexibility can be explained by lengthening the tendon tissues.Nonetheless, the present data provides indirect support for the hypothesis of Fowles et al. (2000) that stretching affects the length-tension relationship which may reduce the muscle's force generating capacity and the theory of changes in mechanical property. The theory of neural adaptation cannot be addressed in the current study, as muscle activation has not been measured in this study.

A conflicting study has also been found in the literature addressing increased peak torque value after stretching (Ozkaya & Nordin, 1999). The conflicting results may relate to changes in antagonist muscle activation; a greater force may develop in the agonist as a result of less resistance to extension movement in the antagonist.

Change in Joint Angle of Peak Torque

In two different studies, it has been illustrated that the changes in the angle at peak torque may relate to that the sarcomeres are producing peak tension at less-than-optimal position (Fowles et al., 2000; Nelson et al., 2001a). Changes in angle of peak torque in isokinetic muscle action have also been reported by Cramer et al., 2004a. Other studies, however, have shown no changes in the angle of peak torque in response to intensive stretching (Cramer et al., 2005; Nelson et al., 2001b).The angle-torque relationship was also investigated in passive isokinetic test. The training program did not significantly affect the passive torque at maximal tolerated and predominated joint angle of knee. Both remained unchanged after 8-week stretching. These results are not in-line with the previously reported studies (Flopp et al., 2006; Magnusson, et al, 1996).

Our findings also indicated that despite the stretching did not significantly change the values of torque at maximal tolerated joint angle, the passive length tension curves became more flat

in the post-test compared to the pre-test; indicating possiblechanges in viscoelastic properties of muscle (Gajdosik, 2001).Therefore, our passive experimental results are inconsistent with the theory of changes in stretch tolerance as the explanation for increased ROM. In the theory of 'increased ROM due to changes in stretch tolerance'(Magnusson et al., 1996c; Halbertsma and Goeken, 1994), it is expected to see no changes in the pre-and post-curves.

Change in Architectural Properties of Hamstring Muscle

The findings of the present study confirm that the stretching did not significantly change the architectural properties of hamstring muscle (fascicle length, FL, and pennation angle, PA). These findingsare consistent with previous studies (Magnusson et al., 1996c; Halbertsma and Goeken, 1994),however it was not in-line with what we expected. In the current study, the intervention period was longer than most previous studies and therefore, changes in muscle architecture was expected.Generally, FL tended to decreaseand the PA became slightly shorter in post assessment compared to the condition before stretching program.

Increased fiber length would limit the loss of fiber force, and also better capacity for developing higher velocities of contraction (Kumagai et al., 2000). An increase of 24.9% in FL was reported by Blazevich et al. (2003) who performed sprint-jump training for 5 weeks.The combination of thicker muscle and longer fascicle would result in greater force output to an identical shortening velocity (Kumagai et al., 2000). Improved force transmission through the fibers is also expected when the pennation angle is decreased (Alegre et al., 2005). Increased FL after stretchingin vivo is only a speculative, but it has been reported in animal muscles (Lynn et al., 1998).

The differences between the current study and those previously reportedmay be attributed to the type of training, limited numberof subjects, and the duration of intervention. Although, no significant change in fascicle length was observed after the stretching program, a right-shift in passive tension curve could also be explained by increased muscle length (Ozkaya & Nordin, 1999). To confirm this, we recommend measuring the subject's maximum tolerated deflection. In the case of increased muscle length, a similar trend in the curve, flatter and right-shift curve at even larger joint angle will be achieved.

Our findings do not support the sensation theory as end-range joint angle of the stretch is unchanged in passive knee at maximal joint angle test. The endpoint of these stretches is subject sensation (Weppler & Magnusson, 2010).

Pain and Tolerance

Pain perception was also measured by visual analog scale (VAS) during passive tests. After 8 weeks of intervention, when pain perception was measured at maximal tolerated joint angle, all the subjects felt the same pain at more stretched position or at more knee joint range of motion; meaning that they could stretch more before they felt the same pain. Therefore, it is tempting to suggest thata stretch program can increase a person's tolerance to stretch. Similarly, altered stretch tolerance following stretch interventions has also been reported Halbertsma&Goeken (1994) and Magnusson et al. (1996). Even though, both groups of authors investigated the effect of stretching on hamstring muscles with relatively intensive regimens of 4 and 3 weeks, respectively, Bjorklund et al. (2001) reported similar results after 2 weeks stretch regime. Therefore, it is not surprising to see similar results after 8 weeks of stretching. The changes in stretch tolerance have been discussed in the literature in connection with nociceptive nerve endings, mechanoreceptors, or proprioceptors (Magnusson et al., 1996; Proske et al., 1993). Alternatively, some other aspect of the sensory neural pathways may alter after stretching (Magnusson et al., 1996; Laessoe & Voigt, 2003); i.e. afferent input from muscles and joints during a stretch may interfere with signals from nociceptive fibers inhibiting an individual's perception of pain (the gate control theory of pain) (Melzack & Wall, 1965). Changes in stretch tolerance have also been explained by psychological contribution. According to this explanation, one may say that the participants may anticipate the positive effects of stretch and, therefore, their perception of pain is dampened; willing to tolerate greater stretch over time (Melzack & Wall, 1965).

The results of passive and also pain perception tests remain somewhat conflicting. The results of passive isokinetic tests did not support the theory of 'changes in stretch tolerance', while pain perception results are in-line with this theory.

Limitations

Several limitations of this study must also be addressed. General ROM was not measured individually for each leg, as the sit and reach test is bilateral. It should be noticed that there are several factors affecting the results of this test; such as movement in pelvis (Gajdosik, 2001), variations in arm, leg and trunk length of the subjects (Walls & Dillon, 1952). Nonetheless, it seems possible that the difference in the pre and post- sit and reach test is related to the unilateral training of hamstring muscle.

Although, subjects were well instructed with respect to training, parts of the training was done on individual basis, and a more controlled intervention would perhaps have ensured a greater compliance to the study intervention.

By the nature of this study, it was not possible to blind a subject as which leg was the intervention leg. It cannot be excluded that somehow personal beliefs regarding the efficacy of stretching may influence performance in the tests.

The present study was as a part of a longer study examining the effects of 24 weeks of stretching, but due to time constraints, the present data only include 8 weeks of stretching. A longer intervention period is likely to yield different or more consistent results.

Gender difference may also influence the results, as there are gender differences in the mechanical and neuromuscular properties of hamstring. Previous studies have confirmed reduced active and passive hamstring stiffness in female participants (Marshall et al., 2009; Blackburn et al., 2009; Granata et al., 2002) and positive relationship between hamstring extensibility and the onset of medial hamstring muscles activation in response to the passive stretch in females.

The subject population is also very important factor affecting the outcome of a research study. This study was completed by totally 7 subjects and more participants may increase the validity of the research study.

Conclusion

In conclusion, the present study confirms increased ROM as a result of 8-week stretching exercise for the hamstring muscles. Moreover, a change in angle of peak torque was observed during active contraction in line with what was expected. Although, the intervention did not result in muscle structural adaptation, and despite no other significant changes were observed in most active or passive contractile parameters it cannot be excluded that the presently observed changes in angle of peak torqueis related to some change in muscle-tendon mechanics/morphology, that perhaps would have been more evident with an even longer intervention period.

Nonetheless, the hypotheses that were put forward initially cannot be confirmed in general with the present data set.

Further study

Further researchis needed to clarify the various mechanisms for increased ROM due to flexibility training.It would have been more evident to measure the subject's maximum tolerated deflection to confirm the increased muscle length after stretching. Although, no changes in muscle architectural properties were observed after the stretching, anobserved right-shift in passive tension curve could also be explained by changes in muscle length (Ozkaya & Nordin, 1999).

Additionally, to obtain a better understanding about the physiological properties of muscle it is essential that other muscle tendon units be examined. The ratio of muscle to tendon may bean important factor when examining the results of stretching. The visco-elasticity of a tendon unit isaltered with stretching, so when a muscle has a long tendon and short muscle, the degree of pennation angle change will be more pronounced compared to the long muscle and tendon short (Fowles et al., 2000; Morse et al., 2008).Therefore, stretching would have differing results on muscles around the body.

Perhaps more comprehensive inclusion criteria shouldbe considered when choosing the subjects. A research study has shown that individuals with different passive torques may have different responses to stretching (Abellaneda et al., 2009).

To check the theory of neural adaptation, muscle activation has to be measured. EMG can be employed for such a purpose.

It would also be interesting to examine the mechanical properties of hamstring tendons. This can be done by ultrasonography, but seems to be difficult with the hamstrings tendons.

The influence of gender on the responsiveness to the stretching intervention is suggested to investigate with sufficient number of subjects. Reduced active and passive hamstring stiffness in female participants have been reported in the literature (Marshall et al., 2009; Blackburn et al., 2009; Granata et al., 2002).

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APPENDICES

Effekt av langvarig bevegelighetstrening – Hoveddel – 14.6.2011

Forespørsel om deltakelse i forskningsprosjektet

”Effekt av 24 ukers bevegelighetstrening på mekaniske egenskaper i muskel-sene-systemet”

Bakgrunn og hensikt
Dette er et spørsmål til deg om å delta i en forskningsstudie for å undersøke hvordan regelmessig bevegelighetstrening påvirker mekaniske egenskaper i muskler, sener og bindevev. Vi håper at studien skal gi oss bedre dokumentasjon på hvilke endringer bevegelighetstrening medfører, og med dét bedre innsikt i hvilke grupper som kan ha nytte av bevegelighetstrening, eventuelt grupper som ikke vil ha utbytte av slik trening. Dette gjelder både ulike grupper av idrettsutøvere, mosjonister og pasienter.

Du forespørres om å delta i studien fordi du tilhører en normal, sunn populasjon, og fordi vi håper at du er interessert i mer kunnskap på dette feltet. Studien og testene som skal gjennomføres er godkjent av regional komité for medisinsk og helsefaglig forskning. Ansvarlig virksomhet er Norges idrettshøgskole (NIH).

Hva innebærer studien?
Hvis du velger å delta i studien, vil du bli trukket ut til å delta i én av to grupper. Treningsgruppen skal gjennomføre et kort bevegelighetstreningsprogram for ankelstrekkerne og hamstringsgruppen, den andre gruppen fungerer som kontroll og gjør derfor ingen bevegelighetstrening. Før og etter treningsperioden foretas en rekke tester av bevegelighet, styrke og funksjon, ved NIH. Det blir også tatt ultralyd og MR av leggen og låret hos alle forsøkspersonene. Av et mindre utvalg av forsøkspersonene vil det bli tatt vevsprøver fra gastrocnemius-muskelen. Se vedlegg A for detaljer om testene.

Mulige fordeler og ulemper
Ved å delta i studien vil du få informasjon om din styrke og bevegelighet i de aktuelle leddene. Om du havner i treningsgruppen, vil du få mulighet til å øke ditt leddutslag betydelig. Du vil få praktisk erfaring med en rekke vitenskapelige tester for bevegelighet og muskelfunksjon, og du vil få god innsikt i hvordan et forskningsprosjekt foregår. Dette kan være nyttig for dine videre studier.

Prosjektet vil kreve en del av din tid. Selve treningsperioden innebærer daglig bevegelighetstrening i 24 uker. Treningsprogrammet tar ca 15 minutter, og er tenkt gjennomført i forbindelse med forelesninger. Programmet er også lagt opp slik at du kan gjennomføre det på egenhånd, på hvilket som helst sted, i helger, ferier og fridager.

Alle forsøkspersoner skal også ta en del tester. Før treningsperioden blir det to tester på NIH, og MR-undersøkelse i Oslo sentrum. Underveis i treningsperioden blir det to tester på NIH. Etter treningsperioden blir det tre tester på NIH, samt MR i Oslo sentrum. Hver test varer mellom 30 og 120 minutter.

Det kan oppleves ubehagelig å gjøre bevegelighetstrening. Du blir oppfordret til å gå til posisjoner som tilsvarer smertegrensen din, men du har til enhver tid full kontroll med hvor hardt du tøyer. Treningsformen som benyttes er skånsom, du vil få god opplæring i øvelsene, og utførelsen din vil jevnlig bli kontrollert av erfarne instruktører, slik at risiko for skader minimeres.

Testene som foretas på NIH måler din bevegelighet, styrke og funksjon. Styrketestene skal gjennomføres med maksimal innsats. Å ta i maksimalt kan oppleves ubehagelig, og kan medføre lett stølhet. Styrketesting kan i sjeldne tilfeller medføre skade på muskulatur eller sener, men risikoen for

dette er ikke større enn ved en aktiv livsførsel. Testprosedyrene anvendes regelmessig i forskning og ved testing av idrettsutøvere.

MR er en skånsom metode der et sterkt magnetfelt sammen med radiofrekvente signaler benyttes for å danne et grafisk bilde av en kroppsdel, i dette prosjektet lår og legg. Metoden medfører intet ubehag eller risiko. Ultralyd gjøres ved at en plastgjenstand – en probe – holdes mot huden din. Proben sender ut lydbølger. Når lyden passerer forskjellige vev, reflekteres litt av lyden tilbake, fanges opp, og omgjøres til bilder i en PC. Det er ikke noe ubehag eller risiko forbundet med slike ultralydundersøkelser.

Et utvalg av forsøkspersonene blir trukket ut til å ta muskelvevsprøver av gastrocnemius-muskelen. Dette kan oppleves ubehagelig, og det er alltid en viss risiko for infeksjon i såret. Erfaringsmessig er infeksjonsfaren svært liten ved de prosedyrer som blir fulgt ved Norges idrettshøgskole, og vi har til dags dato aldri hatt uhell eller skader i forbindelse med muskelvevsprøver. Det tas prøve fra den ene leggen før treningsperioden, den andre etter treningen.

Hva skjer med prøvene og informasjonen om deg?
Prøvene tatt av deg og informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studien. Alle opplysningene og prøvene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger. En kode knytter deg til dine opplysninger og prøver gjennom en navneliste.

Det er kun autorisert personell knyttet til prosjektet som har adgang til navnelisten og som kan finne tilbake til deg. Når resultatene fra prosjektet er ferdig behandlet og prosjektet er avsluttet, vil navnelisten bli slettet, slik at dine resultater ikke kan spores tilbake til deg. Prosjektet planlegges å avsluttes innen utgangen av 2014.

Det vil ikke være mulig å identifisere deg i resultatene av studien når disse publiseres.

Frivillig deltakelse
Det er frivillig å delta i studien. Du kan når som helst og uten å oppgi noen grunn trekke ditt samtykke til å delta i studien. Dette vil ikke få konsekvenser for deg.

Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Om du nå sier ja til å delta, kan du senere trekke tilbake ditt samtykke uten at det vil få konsekvenser for deg.

Dersom du senere ønsker å trekke deg eller har spørsmål til studien, kan du kontakte Marie Moltubakk, telefon +47 90 83 72 27 / +47 23 26 23 23 eller marie.moltubakk@nih.no.

Ytterligere informasjon om studien finnes i kapittel *A – utdypende forklaring av hva studien innebærer.*
Ytterligere informasjon om biobank, personvern og forsikring finnes i kapittel B – *Personvern, biobank, økonomi og forsikring.*

Samtykkeerklæring følger etter kapittel B.

Kapittel A - utdypende forklaring av hva studien innebærer

Kriterier for deltakelse
- Har fylt 18 år, men har ikke fylt 36 år
- Har ingen muskel- eller skjelettsykdommer
- Har ingen skader i ankelen eller i hamstrings i løpet av de siste 6 månedene
- Har ikke drevet regelmessig bevegelighetstrening de siste 5 årene (ikke mer enn 10 min uttøyning, 3 ganger per uke)
- Maksimalt 25° passiv ankel dorsalfleksjon (blir kontrollert ved første oppmøte)
- Kan ikke berøre gulvet når du står med strake knær (blir kontrollert ved "sit and reach" ved første oppmøte)

Bakgrunnsinformasjon om studien
Bevegelighet er en av faktorene som avgjør en persons evne til å løse en gitt bevegelsesoppgave. Bevegelighetstrening benyttes i mange sammenhenger, både med helseperspektiv og innen toppidrett. Det er likevel ikke enighet om hvorvidt bevegelighetstrening kan hindre skader eller forbedre idrettsprestasjoner. Dette har i stor grad sammenheng med at verken mekanismene for endring i bevegelsesutslag eller de ulike mekaniske effektene av bevegelighetstrening er verifisert.

Det finnes et fåtall forskningsprosjekter der mennesker er utsatt for bevegelighetstrening over tid. Disse studiene har typisk lav kvalitet, og/eller en relativt kort treningsperiode. Denne studien er et innledende forsøk på å skaffe mer informasjon om mulige mekaniske effekter av langvarig bevegelighetstrening, for å legge forholdene bedre til rette for fremtidige treningsstudier.

Denne studien undersøker hvordan regelmessig bevegelighetstrening påvirker mekaniske egenskaper i muskler, sener og bindevev. Vi håper at studien skal gi oss bedre dokumentasjon på hvilke endringer bevegelighetstrening medfører, og med dét bedre innsikt i hvilke grupper som kan ha nytte av bevegelighetstrening, eventuelt grupper som ikke vil ha utbytte av slik trening. Dette gjelder både ulike grupper av idrettsutøvere, mosjonister og pasienter.

Tidsskjema – hva skjer og når skjer det?
Hvis du bestemmer deg for å delta i studien, fyller du ut vedlagte svarskjema, og sender det til marie.moltubakk@nih.no. Samtykkeerklæringen på siste side i dette heftet leverer du når du møter til testing.

Når vi har mottatt svarskjemaet ditt, kontakter vi deg for å avtale tidspunkt for første test. Første test finner sted i andre halvdel av september. Etter den første testen får du beskjed om du er kvalifisert til å delta i studien, basert på bevegelighetstester som nevnt over.

I slutten av september/første halvdel av oktober skal du inn til nok en test på NIH, og til MR-undersøkelse i Oslo sentrum. Etter disse testene foretar vi trekningen som avgjør om du skal delta i treningsgruppen eller i kontrollgruppen. Treningsperioden foregår ca oktober-april.

Når treningsperioden er ferdig, rundt påsketider, blir du kalt inn til nye tester på NIH, og ny MR-undersøkelse.

Nøyaktig dato og tidspunkt for testene fastsetter du og forskerne sammen, slik at testingen passer inn i dine øvrige gjøremål.

Undersøkelser som blir gjort av deg

Bevegelighetstester
- Passiv bevegelighet i ankel dorsalfleksjon og hoftefleksjon vil bli testet i et isokinetisk dynamometer. Benet ditt blir festet til dynamometeret, som beveger benet ditt sakte mot ytterstilling. Når bevegelsen har nådd smertegrensen din, trykker du på en knapp. Dette stopper umiddelbart bevegelsen, og fører benet ditt tilbake til nøytral posisjon.
  - o Du kommer til å øve deg på denne testen, uten å gå til smertegrensen, til du er helt komfortabel med prosedyren.
- Passiv bevegelse av ankelen, i et område godt innenfor smertegrensen din, i isokinetisk dynamometer, med ultralyd (se senere).

Styrketester
- Før testene utføres generell og spesifikk oppvarming.
- Kneekstensjon, knefleksjon, ankel dorsalfleksjon og ankel plantarfleksjon testes i isokinetisk dynamometer.
  - o Testene utføres isokinetisk. Det vil si at kneet (eller ankelen) strekkes (eller bøyes) i konstant hastighet, uansett hvor mye eller lite du tar i. Du skal likevel ta i maksimalt.
  - o Testene utføres både konsentrisk og eksentrisk.
- Ankel plantarfleksjon testes i isometrisk dynamometer, med ultralyd (se senere).
  - o I denne testen skal du ta i gradvis, fra lav innsats til maksimal innsats i løpet av 10 sekunder.
  - o Testen utføres isometrisk. Det vil si at maskinen holder ankelen din i samme posisjon, uansett hvor mye du tar i.
- Du kommer til å øve deg på disse testene, uten at du tar i maksimalt, til du er komfortabel med prosedyren.

Hoppetester
- Hinking på ett ben i 30 sekunder, på kraftplattform.
- Maksimale svikthopp på ett ben, på kraftplattform.

EMG
- Små elektroder som måler aktiviteten i musklene dine blir festet på forsiden og baksiden av låret og leggen. Disse elektrodene skal du ha på mens du gjør bevegelighetstestene, styrketestene og hoppetestene.

Ultralyd
- Ultralyd gjøres ved at en plastgjenstand – en probe – holdes mot huden din. Proben sender ut lydbølger. Når lyden passerer forskjellige vev, reflekteres litt av lyden tilbake, fanges opp, og omgjøres til bilder i en PC. Det er ikke noe ubehag eller risiko forbundet med slike ultralydundersøkelser.
- Ultralyd benyttes som et hjelpemiddel i noen av bevegelighets- og styrketestene som er beskrevet over.
- I tillegg tas ultralydbilder av musklene gastrocnemius medialis og biceps femoris, samt av Akillessenen, mens du ligger avslappet på en benk.

MR
- MR er en skånsom metode der et sterkt magnetfelt sammen med radiofrekvente signaler benyttes for å danne et grafisk bilde av en kroppsdel. Metoden medfører intet ubehag eller risiko. Eventuelle årsaker til at man ikke bør ta MR, gjennomgås av personellet før testen.
- MR utføres av spesialutdannet personell hos Curato Røntgen i Oslo sentrum.

- Det gjøres MR av leggen og låret. Ankelen din blir fiksert i 90° vinkel. Du ligger på en benk, og bena dine blir ført inn i en liten tunnel. Undersøkelsen varer ca 20 minutter. Du må ligge helt i ro så lenge undersøkelsen pågår.

Prøver av muskelvev (enkelte forsøkspersoner)
- Atten forsøkspersoner trekkes ut til å ta muskelvevsprøver av gastrocnemius medialis: Ti personer fra treningsgruppen og åtte fra kontrollgruppen.
- Vevsprøver foretas ved hjelp av en spesialnål. Nålen stikkes inn i muskelen, hvor den henter ut en svært liten bit av muskelen. Før prøven gis lokalbedøvelse.
- Det tas vevsprøve fra den ene leggen før treningsperioden, den andre leggen etter treningsperioden.

Antropometriske mål
- Måling av høyde, vekt, benlengde og legglengde.

Spørreskjema
- Dominant ben – hvilket ben foretrekker du å sparke en ball med?
- Treningsbakgrunn
- Nåværende treningsvaner
- Tidligere skader

Mulige fordeler
Ved å delta i studien vil du få informasjon om din styrke og bevegelighet i bena. Om du havner i treningsgruppen, vil du få mulighet til å øke ditt leddutslag betydelig. Du vil få praktisk erfaring med en rekke vitenskapelige tester for bevegelighet og muskelfunksjon, og du vil få god innsikt i hvordan et forskningsprosjekt foregår. Dette kan være nyttig for dine videre studier.

Mulige ubehag/ulemper
Prosjektet vil kreve en del av din tid. Selve treningsperioden innebærer daglig bevegelighetstrening i 24 uker. Treningsprogrammet tar ca 15 minutter, og er tenkt gjennomført i forbindelse med forelesninger. Programmet er også lagt opp slik at du kan gjennomføre det på egenhånd, på hvilket som helst sted, i helger, ferier og fridager.

Alle forsøkspersoner skal også ta en del tester. Før treningsperioden blir det to tester på NIH, og MR-undersøkelse i Oslo sentrum. Underveis i treningsperioden blir det to tester på NIH. Etter treningsperioden blir det tre tester på NIH, samt MR i Oslo sentrum. Hver test varer mellom 30 og 120 minutter.

Det kan oppleves ubehagelig å gjøre bevegelighetstrening. Du blir oppfordret til å gå til posisjoner som tilsvarer smertegrensen din, men du har til enhver tid full kontroll med hvor hardt du tøyer. Treningsformen som benyttes er skånsom, du vil få god opplæring i øvelsene, og utførelsen din vil jevnlig bli kontrollert av erfarne instruktører, slik at risiko for skader minimeres.

Testene som foretas på NIH måler din bevegelighet, styrke og funksjon. Styrketestene skal gjennomføres med maksimal innsats. Å ta i maksimalt kan oppleves ubehagelig, og kan medføre lett stølhet. Styrketestingen kan medføre enkelte skader på muskulatur eller sener, men risikoen for dette er ikke større enn ved en aktiv livsførsel.

Ultralyd og MR av muskel-sene-systemet innebærer intet ubehag eller risiko.

Prøver av muskelvev kan oppleves ubehagelig, og det er alltid en viss risiko for infeksjon i såret. Erfaringsmessig er infeksjonsfaren svært liten ved de prosedyrer som blir fulgt ved Norges idrettshøgskole, og vi har til dags dato aldri hatt uhell eller skader i forbindelse med muskelvevsprøver.

Økonomi
Forsøkspersoner som har lang reisevei til teststedene, vil få mulighet til å levere reiseregning etter nærmere avtale.

Du har rett på informasjon
Alle testene i prosjektet prøves ut i perioden før studiestart. Dersom utprøvingen gir oss informasjon som du bør kjenne til, vil vi gi deg beskjed umiddelbart.

Kapittel B - Personvern, biobank, økonomi og forsikring

Personvern
Opplysninger som registreres om deg er navn, alder, treningsbakgrunn, og resultater fra testene som er beskrevet i kapittel A.

Informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studien. Alle opplysningene og prøvene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger. En tallkode knytter deg til dine opplysninger og testresultater gjennom en navneliste.

Det er kun autorisert personell knyttet til prosjektet som har adgang til navnelisten og som kan finne tilbake til deg. Når resultatene fra prosjektet er ferdig behandlet og prosjektet er avsluttet, vil navnelistene bli slettet, slik at dine resultater ikke kan spores tilbake til deg. Prosjektet planlegges å avsluttes innen utgangen av 2014

Det vil ikke være mulig å identifisere deg i resultatene av studien når disse publiseres.

Norges idrettshøgskole ved administrerende direktør er databehandlingsansvarlig.

Biobank
Muskelvevsprøvene som blir tatt og informasjonen utledet av dette materialet vil bli lagret i en forskningsbiobank ved Norges idrettshøgskole. Hvis du sier ja til å delta i studien, gir du også samtykke til at det biologiske materialet og analyseresultater inngår i biobanken. Jens Bojsen-Møller er ansvarshavende for forskningsbiobanken. Biobanken planlegges å vare til 2014. Etter dette vil materiale og opplysninger bli destruert og slettet etter interne retningslinjer.

Utlevering av materiale og opplysninger til andre
Hvis du sier ja til å delta i studien, gir du også ditt samtykke til at prøver og avidentifiserte opplysninger utleveres til Bispebjerg Hospital, Danmark, som er samarbeidspartner i prosjektet.

Rett til innsyn og sletting av opplysninger om deg og sletting av prøver
Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har videre rett til å få korrigert eventuelle feil i de opplysningene vi har registrert. Dersom du

trekker deg fra studien, kan du kreve å få slettet innsamlede prøver og opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner.

Økonomi og Norges idrettshøgskoles rolle
Studien og biobanken er finansiert gjennom midler fra Norges idrettshøgskole. Denne finansieringen innebærer ingen interessekonflikter, etiske eller praktiske utfordringer.

Forsikring
Deltagere i studien er forsikret gjennom Norges idrettshøgskoles næringslivsforsikring hos Gjensidige.

Informasjon om utfallet av studien
Som deltager i prosjektet har du rett til å få opplyst både dine egne resultater, og informasjon om resultatene av studien totalt sett. Denne informasjonen vil bli sendt til forsøkspersonene når prosjektet avsluttes. Du kan også få tilsendt informasjonen ved å kontakte marie.moltubakk@nih.no.

Samtykke til deltakelse i studien

Jeg er villig til å delta i studien

(Signert av prosjektdeltaker, dato)

Stedfortredende samtykke når berettiget, enten i tillegg til personen selv eller istedenfor

_____

(Signert av nærstående, dato)

Jeg bekrefter å ha gitt informasjon om studien

(Signert, rolle i studien, dato)

Forsøksperson-nummer: _____ Dato: _____ Klokkeslett: _____

Standardisering av test: Dynamometer   MR   Biopsi

Trening de to siste dagene før test:

NB, unngå hard styrketrening de to siste dagene!

Måltider i forkant av testen (tidspunkt, innhold og mengde):

Treningstøy og sko på testen:

For jenter: Første dato for forrige menstruasjon, evt. om du bruker p-piller.

Hvilke idretter/treningsformer har du drevet med de siste 3 årene?

Ca hvor mange TIMER trener du pr uke, gjennomsnitt for høsten 2011?   [   ]

Ca hvor mange MINUTTER trener du bevegelighet pr uke, gjennomsnitt for høsten 2011?   [   ]

Med hvilket ben foretrekker du å sparke en ball?   ☐ Høyre   ☐ Venstre

---

Beskriv eventuelle skader du har/har hatt i bena de siste 3 årene (type skade, hvilken periode, og

om du har fått behandling for skaden):

Oppvarming 1: Diagonale hopp

Raske hopp til diagonal stilling, vekselvis høyre og venstre ben.

Viktig!   ● Foten må peke rett forover
            ● Hælen må være i gulvet

Antall rep: 30 repetisjoner på hver side = totalt 60 hopp.

Oppvarming 2: Jumping Jacks

2 raske hopp til lateral stilling + 1 hopp til 90 graders knebøy.

Viktig!   ● Knær og føtter i naturlig utoverrotert stilling

Antall rep: Antall rep: 3 hopp x 10 repetisjoner = totalt 30 hopp.

Oppvarming 3: Diagonale utfall

1 utfall forover med LETT bøyd kne, strekk armene mot foten.
Deretter 1 utfall bakover med strakt kne, sett hælen i gulvet.
Øvelsen utføres KUN på det benet som skal tøyes

Viktig! • Utfall forover: Kneet må være strakt nok til at du kjenner lett tøyning av hamstr.
• Parallell fotstilling gir deg bedre balanse
• Bevegelsen skal være kontrollert. Etter hvert som kontrollen øker,
  kan du øke tempoet.

Ant rep: 8 i hver retning = totalt 16 utfall.

Oppvarming 4: Laterale utfall

1 utfall til siden, LETT bøyd kne, strekk armene mot foten. Deretter 1 utfall
der du krysser bena, LETT bøyd kne, strekk armene mot lilletåsiden av foten.
Øvelsen utføres KUN på det benet som skal tøyes

Viktig! • Kneet må være strakt nok til at du kjenner lett tøyning av hamstrings
• Kne og fot i naturlig rotert stilling
• Bevegelsen skal være kontrollert. Etter hvert som kontrollen øker,
  kan du øke tempoet.

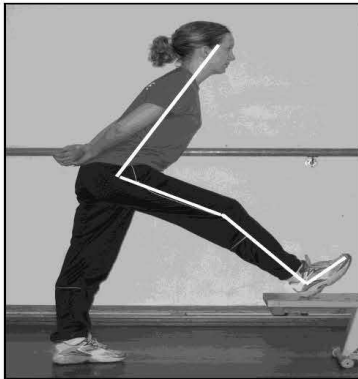**Ant rep:** 8 i hver retning = totalt 16 utfall.

Hamstrings, passiv tøyning

Stå med det ene benet på en benk, trappetrinn, ribbevegg e.l. Lett bøyd kne, nøytral posisjon i ankelleddet. Bøy strak overkropp forover mot foten.

Viktig!   ● Strak rygg
              ● Lett bøyd kne

Holdetid: 1 min
Antall rep: 4

Utfylling:

Hver uke har en egen linje, hver ukedag har sin egen kolonne (loddrett). Hver ukedag er igjen delt inn i ankel og hamstrings. På denne måten kan du markere hvis du ikke har fått utført alle øvelsene pga. skade e.l.

Hvis du har vært syk, har fått en skade eller annet som gjør at treningen ikke har gått som normalt, skal dette føres opp under merknader.

EKSEMPLE TRENINGSLOGG FOR:

		M	Ti	O	To	F	L	S	Marknader
UKE 3	hamstrings (4 x 1 min)	✓	✓	✓	✓	✓	✓	✓	
UKE 4	hamstrings (4 x 1 min)	✓	✓			✓	✓	✓	Hadde høy feber ons og tors og fikk ikke trent.
UKE 5	hamstrings (4 x 1 min)	✓	✓	✓	✓				strekk på fotballtreninga.
UKE 6	hamstrings (4 x 1 min)	✓	✓	✓	✓	✓	✓	✓	
UKE 7	hamstrings (4 x 1 min)	✓	✓	✓	✓	✓	✓	✓	
UKE 8	hamstrings (4 x 1 min)	✓	✓	✓	✓	✓	✓	✓	
UKE 9	hamstrings (4 x 1 min)	✓	✓	✓	✓	✓	✓	✓	
UKE 10	hamstrings (4 x 1 min)	✓	✓	✓	✓	✓	✓	✓	

TRENINGSLOGG FOR:

		M	Ti	O	To	F	L	S	Marknader
UKE 3	hamstrings (4 x 1 min)	☐	☐	☐	☐	☐	☐	☐	
UKE 4	hamstrings (4 x 1 min)	☐	☐	☐	☐	☐	☐	☐	
UKE 5	hamstrings (4 x 1 min)	☐	☐	☐	☐	☐	☐	☐	
UKE 6	hamstrings (4 x 1 min)	☐	☐	☐	☐	☐	☐	☐	
UKE 7	hamstrings (4 x 1 min)	☐	☐	☐	☐	☐	☐	☐	
UKE 8	hamstrings (4 x 1 min)	☐	☐	☐	☐	☐	☐	☐	
UKE 9	hamstrings (4 x 1 min)	☐	☐	☐	☐	☐	☐	☐	
UKE 10	hamstrings (4 x 1 min)	☐	☐	☐	☐	☐	☐	☐	

Subject number: **Date:**   **Time:**

Basic data:	Height ___ cm		
	Weight ___ kg		
	Sit-and-reach: ___ cm (negative=inflex)		

Chair settings KNEE:			
chair front/back _____		column front/back _____	
column height _____		moment arm _____	

Dynamometer, passive resistance KNEE: 2x isokinetic to subject endpoint				**Dyno** f991pkmr / l
flex24pkm	maximal end ROM	R: ___	L: _____	
max-75°	peak torque	R: ___	L: _____	

Dynamometer, passive resistance KNEE: 2x isokinetic to submax endpoint				**Dyno** f991pkpr / l
flex24pkp	predetermined ROM	R: ___	L: _____	
(max-10)-75°	peak torque	R: ___	L: _____	

Dynamometer, active torque KNEE: dorsi 3+3w + 4 test, plantar 3+3w + 4 test				**Dyno** f991akcr / l
flex24akc	predetermined ROM	R: ___	L: _____	
(max-10)-75°	extension peak torque	R: ___	L: _____	
	ext angle of peak torque	R: ___	L: _____	
	flexion peak torque	R: ___	L: _____	
	flex angle of peak torque	R: ___	L: _____	

File Names:

Subjects:	f99r/l
Test Times:	1= fam, 2=pre, 3=8wk
Passive/ Active:	p/a
Mode:	m=Maximal Passive, p= Predefined Passive, c=Concentric, u=Ultrasound
Side:	r/l