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TITLE PAGE

Stretching: The mechanisms and benefits for sport performance and injury prevention.

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Running title

The mechanisms and benefits of stretching

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ABSTRACT

Stretching is usually performed before exercise in an attempt to enhance performance and reduce the risk of injury. Most stretching techniques (static, ballistic, and proprioceptive neuromuscular facilitation) are effective in increasing static flexibility as measured by joint range of motion, but the results for dynamic flexibility as measured by active and passive stiffness, are inconclusive. The mechanisms of various stretching techniques in terms of biomechanics and neurology, the effectiveness of the combination of stretching with other therapies such as heat and cold, and the effectiveness of stretching for performance and injury prevention, are discussed. The possible mechanisms responsible for the detrimental effects of stretching on performance and the minimal effects on injury prevention are conferred, with the emphasis on muscle dynamic flexibility. Further research is recommended to explore the mechanisms and effects of other stretching techniques besides static stretching on dynamic flexibility, muscle soreness, sport performance, and rate of injury.

INTRODUCTION

Common clinical practices suggest that pre-exercise stretching can enhance performance and prevent injuries by increasing flexibility. However, current scientific research does not support this notion.[1-3] Rather the acute effects of stretching can have detrimental effects on performance parameters such as muscle strength,[2, 4, 5] and jumping performance.[6, 7] In this paper, the mechanisms of stretching are reviewed in order to provide guidelines regarding use of stretching as an appropriate strategy to enhance performance and reduce the risk of injury. The effects of stretching on performance and muscle soreness are presented. Further areas for research are also recommended.

Literature for this review was located using three electronic databases (PubMed, SPORT Discus, and ProQuest 5000 International) in addition to manual journal searches. The computer databases provided access to biomedical and sport-oriented journals, serial publications, books, theses, conference papers, and related research published since 1965. The key search terms included: sport stretching, static stretching, dynamic stretching, ballistic stretching, proprioceptive neuromuscular facilitation, performance, sport injury, delayed onset muscle soreness, injury prevention, and muscle stiffness. There were a limited number of published randomised controlled trials, therefore, other types of research such as clinical controlled trials and literature reviews were included in this review. Articles not published in English and/or in scientific journals, that focused on the psychological effects of stretching, or the effects of stretching in special populations were not included in this review. The criteria for inclusion were that the the article must have:

- focused on normal, healthy participants. Age, gender, and fitness differences were not excluding factors.

- investigated the acute effects of stretching. Immediate and long-term effects of flexibility training were not excluding factors.
- discussed the possible mechanisms of stretching in relation to biomechanical and/or neuromuscular properties of muscle, sport performance, rate of injury, or muscle soreness.

DEFINITION OF STRETCHING

Several literature reviews have considered flexibility[8, 9] as the outcome of stretching exercise. However, the definition of stretching itself has not been well defined yet. Magnusson et al.[10] stated that "stretching has been characterised in biomechanical terms in which the muscle-tendon unit is considered to respond viscoelasticity during the stretching manoeuvre" (pp. 77). However this definition of stretching is more a biomechanical result of stretching rather than a definition of the action of stretching. In our review stretching is defined by the authors as movement applied by an external and/or internal force in order to increase muscle flexibility and/or joint range of motion. The aim of stretching before exercise is to increase muscle-tendon unit length[11] and flexibility. The increase in flexibility may help to enhance athletic performance and decrease the risk of injury from exercise.[9]

Types of stretching technique

There are various types of stretching techniques that are used, often depending on athlete choice, training programme, and the type of sport. An earlier review of stretching[8] indicated that four different methods are commonly used for sport activities: static, ballistic, proprioceptive neuromuscular facilitation (PNF), and dynamic (see Table 1).

Insert Table 1 about here.

MECHANISMS OF STRETCHING

Stretching results in elongation of muscles and soft tissues through mechanical and neurological mechanisms. Stretching activities may benefit athletes mentally through psychological mechanisms, however, there have been no studies on the psychological effects of stretching.

Biomechanical mechanisms

Muscle-tendon units can be lengthened in two ways: muscle contraction and passive stretching. When muscle contracts, the contractile elements are shortened, while the entire length of muscle-tendon unit is fixed. Therefore, a compensatory lengthening occurs at the passive elements of tissues (tendon, perimysium, epimysium, and endomysium).[19] When muscle is lengthening, the muscle fibres and connective tissues are elongated because of the application of external force.[19] Stretching increases muscle-tendon unit length by affecting the biomechanical properties of muscle (range of motion and viscoelastic properties of the muscle-tendon unit).

Range of motion

The majority of previous research on the effects of stretching on flexibility used range of motion as an indicator.[15, 18, 20-22] The exact physiological mechanism of stretching resulting in increased range of motion still remains unclear[23] as most research has failed to show changes in muscle properties such as passive[24-29] or active[30, 31] stiffness. The increase in

range of motion is thought to be the influence of increasing stretch tolerance[18, 24-26, 32] and pain threshold, or subject bias following an intervention.[33] Therefore, the increase in static flexibility, as indicated by range of motion, does not provide clear information on musculotendinous behaviour.[24-26]

Viscoelastic properties of the muscle-tendon unit

The viscoelastic properties of muscle exhibit several phenomena when external load is applied. When tissues are held at a constant length, the force at that length gradually declines and is described as the "stress relaxation" response.[24-26, 33-35] When tissues are held at a constant force, the tissue deformation continues until approaching a new length and is termed "creep".[11] Creep might be another explanation for the immediate increased range of motion after static stretching.[32] The musculotendinous unit also produces a variation in the load-deformation relationship between loading and unloading curves.[11] The area between the loading and unloading curves is termed "hysteresis" and represents the energy loss as heat due to internal damping.[11, 36] Several researchers have studied the effects of stretching on stress-relaxation, creep, and hysteresis,[11, 18, 19, 24-26, 33, 35-39] however, none of the previous research clearly showed the relationship of these phenomena to the rate of muscle injury or performance.

Passive stiffness refers to the resistance of the muscle-tendon unit when external forces are applied. The slope of the force and deformation curve at any range of motion is defined as passive stiffness.[9, 24] Passive torque, which occurs during passive movement, is resistance from stable cross-links between actin and myosin, non-contractile proteins of the endosarcomeric and exosarcomeric cytoskeletons (series elastic components), and connective tissues surrounding muscles (parallel elastic component).[32] Perimysium is considered to produce major resistance.[25, 26] Active stiffness is defined as the ability to transiently deform

the contracted muscle,[9] and can be measured by the damped oscillation technique.[31, 40, 41] The oscillation of the contracted muscle after the application of external force results from the viscoelasticity of muscle and the level of muscle activation.[31] Passive and active stiffness provide more information on muscle-tendon unit behaviour during movement than range of motion only.

Neurological mechanisms

Biomechanical responses of muscle–tendon units during stretching are independent of reflex activity[11, 25, 28, 34, 42] as indicated by the lack of muscle activity (EMG) responses during stretching. However, a decrease in the Hoffman reflex response (H-reflex) during[43] and after stretching[44-47] has been reported.

Some research reports have stated that all stretching techniques affect neural responses by reducing neural sensitivity.[43-48] The majority of research on the effects of stretching on neurological mechanisms have investigated the changes of the H-reflex - the electrical analogue of the stretch reflex but without the effects of gamma motoneurons and muscle spindle discharge.[49] Electrical stimulation of a mixed peripheral nerve (both sensory and motor axons)[49] will evoke the H-reflex. The activation of the motor axons directly induces the M-wave (from the point of stimulation to the neuromuscular junction) prior to evoking the H-reflex (from Ia afferents arising from annulospiral endings on the muscle spindle) via a monosynaptic connection to the alpha motoneurons.[49] H-reflex is widely used to study changes in the reflex excitability of a group of muscle fibres.[43, 46, 47, 49-52] The depressed amplitude of H-reflex after stretching might be due to several possibilities relating to presynaptic and/or postsynaptic change.[53] The presynaptic changes might be due to a presynaptic inhibition inducing an autogenic decrease in Ia afferents and/or an altered capacity for synaptic transmission during repetitive activation.[53] The postsynaptic changes

might be due to an autogenic inhibition from the Golgi tendon organ (GTO), recurrent inhibition from the Renshaw loop, or postsynaptic inhibition of afferents from joint and cutaneous receptors.[53]

Mechanisms of each stretching technique

Even though each technique of stretching is expected to increase muscle and joint flexibility, different stretching techniques produce increases in flexibility by different mechanisms.

Static stretching

Static stretching is the most widely used technique by athletes due to its simplicity. Static stretching was found to affect both mechanical[1, 25, 26, 28, 33-36, 38] and neurological[43-45, 53, 54] properties of the muscle-tendon unit resulting in increased musculoskeletal flexibility (see Table 2).

Insert Table 2 about here.

Despite static stretching being effective in increasing static flexibility as measured by range of motion,[18, 25, 26, 31, 35] static stretching does not affect dynamic flexibility as measured by passive[18, 24, 35] or active[30, 31] stiffness, but affects viscoelastic properties by reducing stress relaxation.[18, 23, 25, 26, 33, 35] The reduction of stress relaxation is an acute adaptation of the parallel elastic component to lower the imposed load across the myotendinous junction where injury usually occurs.[25, 26] However, there is no clear evidence that static stretching can reduce the rate of injury. Static stretching has produced similar muscle-tendon unit property

responses between neurologically intact participants and spinal cord-injured participants with complete motor loss.[26] There were reports of no EMG activity during passive stretching.[25, 26, 28, 33, 34] Therefore, the effects of static stretching on muscle properties do not involve the neurological mechanism.

The effects of stretching on muscle properties depend on various factors including the stretching techniques used, time to stretch, holding duration, time to rest, and the time gap between intervention and measurement. The majority of research has examined the acute effects of static stretching on passive properties of the muscle-tendon unit.[24-26, 33, 35, 39] In a series of studies by Magnusson et al.[25, 26, 33, 56] static stretching at 90 s for five repetitions reduced muscle resistance measured by passive stiffness, peak torque, and stress relaxation. The decline of muscle-tendon unit resistance returned to baseline within one hour[25, 26] except for stress relaxation.[33] Unfortunately, shorter stretch holding times (less than 60 s), and lower stretching repetitions (less than four times) did not provide such effects.[18, 35, 39] Interestingly, long term training using ten stretches for 45 s per day (three weeks)[25] and four stretches for 45 s, two sessions per day, seven days per week for 13 weeks[29] did not change the mechanical or viscoelastic properties of muscle.[25] Therefore, the changes of viscoelasticity of muscle-tendon units depend more on the duration of stretch rather than the number of stretches[36] or the length of the stretching training period. Indeed, if a decrease in muscle-tendon unit resistance is required long stretch duration (up to 90 s) might be most beneficial.

Prolonged static stretch (five to ten minutes) has been shown to decrease tendon and aponeurosis stiffness (elasticity) and hysteresis (viscosity) as measured passively by ultrasonography.[36, 38] The decrease in stiffness from stretching may be due to an acute change in the arrangement of collagen fibres in tendon.[36] However, the holding time in the Kubo et al.[36, 38] studies is considered very long when compared with practical stretching (30-60 s hold). Unfortunately, no research on the effects of static stretching on the tendon for shorter

durations has been identified in the published literature. The same group of researchers[38] also reported that the combination of long term resistance (70% of one repetition maximum, 10 repetitions per set, five sets per day, four days per week for eight weeks) and stretching (ten minutes per day, seven days per week for eight weeks) training did not change tendon elasticity (determined by stiffness) but reduced hysteresis (17%). Unfortunately, the mechanisms responsible for the decrease in stiffness and hysteresis of the tendon are still unknown. The acute response of stretching on tendon and aponeurosis stiffness might be partly responsible for the increase in range of motion.

The effects of stretching on series elastic components are also unclear. The findings of several researchers are in agreement that static stretching does not affect active stiffness. McNair and Stanley[31] and Hunter et al.[55] suggested that soleus stretching (five stretches of 30 s hold and 30 s rest, and 10 stretches of 30 s hold, respectively) did not reduce active stiffness. Similarly, Cornwell and Nelson[30] reported that stretching did not affect the active stiffness of musculotendinous units of triceps surae. Unfortunately, Cornwell and Nelson[30] did not report the details of the research process such as the stretching technique nor active stiffness measurement. Recently, Cornwell et al.[1] found a slight but significant reduction of active muscle stiffness of the soleus muscle (2.8%) after stretching (six stretches of 30 s hold and 30 s rest). McNair and Stanley[31] and Hunter et al.[55] only investigated the soleus muscle while Cornwell et al. investigated the triceps surae. Stretching in Cornwell et al.'s study was slightly longer than in McNair and Stanley's study (180 s and 150 s, respectively) but not longer than Hunter et al.'s study (300 s). In the McNair and Stanley and Hunter et al. studies, the participants stretched the plantar flexors by adopting a step-standing position and stretching by flexing both knees. The participants in the Cornwell et al. study stretched in two ways. The participant placed the foot on the inclined board and maximally dorsiflexed the ankle joint whilst keeping the sole of the foot flush with the board surface and the knee joint

fully extended. The second stretching technique employed the same protocol but the knee was flexed in order to increase the stretching force on the soleus. These stretching protocols might provide more strain on series elastic components of muscle than the method used in the McNair and Stanley study.

In evaluating the effects of long-term stretching, Wilson et al.[40] reported that flexibility training of pectoralis and deltoid muscles (10 to 15 minutes per session, twice per week for eight weeks) reduced active muscle stiffness by 7.2%. The decrease in active stiffness might be from the long-term adaptation of connective tissue, sarcomere, contractile tissue, and/or reflex responses. These effects may not occur with acute stretching.

Despite the fact that the neurological mechanism is not responsible for the change of muscle-tendon unit properties during passive stretching, static stretching has been reported to decrease neuromuscular sensitivity as indicated by H-reflex responses.[43, 44, 53] Rosenbaum and Henning[45] reported that a static stretch of triceps surae (30 s each for three times) reduced the peak force of reflex force production, force rise rate, and EMG activity. Stretching might improve muscle compliance (reduced peak force and force rise rate), reduce muscle spindle sensitivity (reduced peak-to-peak amplitude), and reduce excitation-contraction coupling (increased force-to-EMG ratios). Thigpen et al.[48] proposed that the decrease of evoked H-wave amplitude might be due to inhibitory effects of the Ib afferent from the GTO. Avela et al.[44] also reported a reduction of H-reflex (46%) after prolonged stretching for one hour. The reduction of stretch reflex activity (reduced peak-to-peak amplitude) and α-motoneuron pool excitability (reduced H-wave /M-wave ratio) were suggested to occur from reduced sensitivity of the large-diameter afferents. Vujnovich and Dawson[43] compared two stretching techniques (static and ballistic stretching) on the excitability of the α-motoneurons as indicated by amplitude changes in the H-reflex. H-reflex was reduced by up to 55% during static

stretching of the soleus muscle (maintained for 160 s) but returned to baseline immediately following the termination of stretching. The amplitude of stretching (mid- and full-range of motion) provided a similarity in the mean depression of the H-reflex amplitude. Therefore, the receptor that is likely to mediate the inhibitory effect during static muscle stretch is the muscle spindle type II afferent. The results of this study were questioned because there were only two participants in the mid-range of motion stretching group. In contrast, Guissard et al.[53] reported that greater stretching amplitude produced a greater reduction of H-reflex. When the ankle was moved passively for 10°, the H-reflex reduced by 25% but when the ankle was moved up to 20°, the H-reflex was reduced by 54%. Each stretch was held for 20 s to 30 s. The results from Guissard et al.'s study showed that a reduction of motoneuron excitation during stretching resulted from both pre-and post-synaptic mechanisms. The pre-synaptic mechanism was responsible for the small stretching amplitude while postsynaptic mechanisms played a dominant role in larger stretching amplitudes.

Insert Table 3 about here.

A reduction of neuromuscular sensitivity during stretching, such as the amplitude of the H-reflex, might be due to the tension on stretched muscle applied by an external force being higher than the resistance from a protective mechanism of muscle from a stretch reflex. When muscle is stretched further, neuromuscular excitability from the stretch reflex still works but cannot resist the external force, which might cause the reduced sensitivity of the muscle spindles and, consequently, H-reflex during stretching. A decreased neuromuscular sensitivity, as indicated by the amplitude of the H-reflex, however, reverted to the control

levels immediately after termination of static stretching in a study by Vujnovich and Dawson.[43] Therefore, the neuromuscular mechanism is not likely as the mechanism to increase muscle flexibility after static stretching.

Ballistic stretching

Ballistic stretching is likely to increase flexibility through a neurological mechanism. The stretched muscle is moved passively to the end range by an external force or agonist muscle. Holding a muscle at the end range of joint motion might reduce muscle spindle sensitivity, with repeated stretch applied at the end range inhibiting the GTO. Research has reported an increase in range of motion,[43, 57] decrease in EMG[58] and decrease H-reflex[43] with ballistic stretching.

Only one study in the published literature[43] examined the effects of ballistic stretching on neuromuscular excitability. Ballistic stretch applied following static stretch demonstrated lowered H-reflex mean amplitude than that obtained during static stretching.[43] The lower H-reflex might be due to the inhibition of GTO or presynaptic inhibition from type Ia afferents. The results should be interpreted with caution as the number of subjects in the static stretching and ballistic stretching groups were different (n = 14 and 5 respectively) and there was no control group. Ballistic stretching was performed immediately after static stretching, therefore, the effects of ballistic stretching alone on H-reflex are still unknown.

It has been suggested that ballistic stretching may be more harmful than other stretching techniques. During ballistic stretching, muscle is stretched at a fast rate and rebounded back repetitively, resulting in greater tension and more absorbed energy within the muscle-tendon unit.[11] Muscle, which is released immediately after applying a high force, does not allow enough time for muscle to reduce tension (stress relaxation) or increase length (creep).[11] Surprisingly, scientific evidence did not support this suggestion that ballistic stretching is

more harmful than static stretching. Ballistic stretching (60 bounces per minute, 17 stretches per set for three sets) resulted in less severity of muscle soreness than static stretching (the same intensity and duration, but static stretching was held for 60 s) in college-age male volunteers.[13] Despite ballistic stretching being less harmful than static stretching, according to Smith et al.,[13] most researchers still recommend slow static stretch before exercise.[8, 17]

Proprioceptive Neuromuscular Facilitation (PNF)

Several PNF techniques have been used to increase flexibility including slow-reversal-hold, contract-relax, and hold-relax techniques.[8] These techniques include the combination of alternating contraction and relaxation of both agonist and antagonist muscles.[8] The theory of PNF has been discussed and reviewed recently.[14] The contractility property of muscles provides flexibility in the PNF technique on the basis of the viscoelastic characteristics of muscle and neuromuscular facilitation. The contracted muscle results in lengthening non-contractile elements (perimysium, endomysium, tendon) of muscle, and consequently, causes a relaxation of the muscle–tendon unit and decreased passive tension in a muscle.[19] The contracted muscle also stimulates the muscle sensory receptors within the muscle-muscle spindle (negative stretch reflex) and GTO which help to relax the tensed muscle. Therefore, the muscle-tendon-unit becomes more relaxed after the contraction.

Some PNF techniques, such as slow-reversal-hold, require agonist muscle to contract in order to relax antagonist muscle.[8] "Reciprocal inhibition" occurs when the excitability signal from agonist muscle is transmitted by one set of neurons in the spinal cord to elicit muscle contraction, and then an inhibitory signal is transmitted through a separate set of neurons to inhibit the antagonist muscle.[14] Reciprocal inhibition helps all antagonistic pairs

of muscle to make smooth movement. When antagonist muscle is inhibited, muscle will be stretched to the opposite direction more easily.

Isometric contraction is commonly performed prior to passive stretching in the PNF technique. Post-isometric contraction exhibited a brief decrease in H-reflex response (83% by one second and 10% by ten seconds, respectively).[47] The depressed H-reflex was regardless of the intensity of isometric contraction,[59] velocities, and amplitude of stretch.[46] Researchers have proposed that the decrease in H-reflex after isometric contraction could be a result of pre-synaptic inhibition.[46, 47] The suppression of reflex activity was short-lasting (less than 10 s), indicating that passive stretching should be performed immediately after pre-isometric contraction in order to gain the maximal efficiency of stretching.

PNF stretching has been reported to result in a greater improvement of range of motion compared with static stretching.[25, 26, 60] Post-isometric contraction reduced neuromuscular sensitivity[46, 47] and might help to enhance the effectiveness of stretching (see Table 4). Toft et al.[61] compared short-term (90 minutes after stretching) and long-term (three weeks) effects of contract-relax stretching (maximal contraction of plantarflexors for eight seconds, relaxation for two seconds, and passive stretch for eight seconds) on stress relaxation of ankle plantarflexors. There was no difference between short-term and long-term effects of PNF stretching. In the studies by Toft et al.[61] and McNair et al.,[35] peak torque of the plantarflexors sixty seconds after the start of the stress-relaxation phase was reduced by approximately 15% by PNF stretching,[61] and 20% by static stretching.[35] Similarly, peak torque of the hamstrings declined 18% after PNF stretching and 21% after static stretching in other studies by Magnusson et al.[25, 26] If dynamic flexibility is required, the claim that PNF stretching provides a better flexibility technique than static stretching is still questioned.

Insert Table 4 about here.

PNF stretching is a complicated stretching technique with a combination of shortening contraction and passive stretching. Therefore, PNF stretching might be harmful as it has been found to increase blood pressure[62] and EMG activity[63] during the contraction phases. Moreover, PNF technique needs some experience to be performed, and a partner is needed to help with stretching. When compared with other stretching techniques, the selection of PNF stretching prior to exercise is still being questioned. Further study is required to investigate the effects of PNF stretching on dynamic muscle properties (e.g., active and passive stiffness), performance, and muscle soreness.

Dynamic stretching

In an extensive review of warm-up and stretching,[8] it was stated that "dynamic stretching is important in athletic performance because it is essential for an extremity to be capable of moving through a non-restricted range of motion" (pp.272). Unfortunately, there was no published research cited for any aspects of dynamic stretching.

Cyclic stretching, or passive continuous motion, has been demonstrated to be effective for decreasing passive muscle stiffness.[35] A less stiff muscle is believed to absorb greater energy when forces are applied to it.[35] As well, less muscle stiffness might be beneficial in reducing the severity of muscle soreness as research has shown the positive relation between passive stiffness and the severity of muscle soreness.[64] However, there is no evidence that dynamic movement of stretching can reduce the severity of muscle soreness.

Dynamic stretching might be a useful protocol for increasing flexibility without decreasing athletic performance. Dynamic contraction of muscle throughout the range of motion is expected to decrease dynamic flexibility as indicated by passive muscle stiffness.[35]

Movement, without holding at end range of motion, may not reduce neuromuscular sensitivity. If the effect of decreasing passive stiffness, however, is more pronounced than the effects of neuromuscular sensitivity on performance, a reduction in performance might still occur. Further research is needed to elucidate the benefits of dynamic stretching on flexibility, muscle properties, neuromuscular sensitivity, performance, and injury prevention.

COMBINATION OF STRETCHING WITH OTHER THERAPIES

Warm-up

Stretching is generally performed after warm-up.[8] The theory is that warm-up will increase muscle temperature to help enhance tissue flexibility.[39] Warm-up has been shown to increase tissue temperature but not to affect muscle properties (passive energy absorption) during stretching.[39] In a study by Magnusson et al.,[39] warm-up (jogging) was performed at 70% of maximum O_2 uptake for 10 minutes and resulted in elevation of muscle temperature by 3°C. After warm-up, four static stretch manoeuvres of 90 s reduced passive energy absorption by 25% while five static stretching exercises (held for 90 s) at resting temperature (no warm-up) reduced passive energy absorption by 30%.[25, 26] However, there were no data on other parameters of passive muscle properties such as passive muscle stiffness and peak torque collected in this study. In the same way, warm-up (running[65] and heel raising[66]) did not help to improve range of motion of lower limbs regardless of the warm-up intensity (60, 70, and 80% of VO_2 max)[65] or training period (two, four, and six weeks).[66] Therefore, warm-up might not be an effective way to enhance flexibility of passive properties of muscle.

McNair and Stanley[31] reported the effects of warm-up (treadmill jogging for 10 minutes at 60% of maximum age predicted heart rate) on series elastic muscle stiffness. Surprisingly, warm-up reduced active (series elastic component) stiffness (6%) more than the combination

treatments of warm-up and stretching (3%) and stretching alone (-1%). Warm-up might be more effective in reducing resistance from various properties associated with active stiffness (passive joint properties, level of muscle activation, tendon properties, and the effect of stretch reflex) than stretching.

Warm-up might help to increase muscle relaxation by reducing EMG activity. Mohr et al.[42] reported that post-warm-up EMG activity was significantly less than pre-warm-up EMG activity in gastrocnemius and soleus muscle. The authors proposed that muscle architecture and arrangement of connective tissue might influence the effects of warm-up (cycling) on reducing EMG activity of the gastrosoleus complex. EMG during stretching in the Mohr et al.[42] study was slightly higher than previous reports[24, 27, 33, 35, 39, 67] due to this study using needle electrodes and participants stretching by themselves (weight-bearing position). Participants in previous research were investigated for EMG activity by using surface electrodes and were passively stretched in a relaxed position. Therefore, an application of warm-up prior to stretching in order to reduce EMG activity might be important in some muscles (i.e., gastrocnemius and soleus) which are dense in connective tissue, and with some challenging stretch positions that need more control and balance of the body such as a standing bent knee stretch or a standing straight knee stretch with heel overhanging a step.

Heat and cold

Temperature has effects on muscle[68] and connective tissue[69] in vivo. In a study of skeletal muscle tensile behaviour, warm muscle (40 °C, measured using an intramuscular probe) showed less stiffness and more load-to-failure than cold (35 °C) muscle.[68] A study of rat tail tendon indicated that an elevated tissue temperature (45 °C), before the application of low force (one-quarter of full load to failure), produced greater residual length and reduced

tissue damage (indicated by tissue rupture which was defined as the point at which elongation continued with no increase in load) than tissues with a normal temperature.[69] The results of these animal studies[68, 69] support the general practice by therapists of applying superficial heat before stretching in order to maximise the effectiveness of treatment.

In human, results of research on the combined treatment of stretching with either heat or cold on flexibility as measured by range of motion were inconslusive.[20, 70, 71] In the study by Henricson et al.,[20] application of an electric heating pad (43°C) for 20 minutes before PNF stretching significantly increased range of motion of hip flexion and abduction. The stretching only group increased range of motion of hip flexion and external rotation, while the heat only group did not show any effect on range of motion. The stretching in this study was performed in only one direction (hip flexion) while the participants were investigated for hip flexion, abduction, and external rotation. Taylor et al.[70] compared the effects of static stretch alone (held for one minute), heat (77°C) and static stretch, and cold (-18°C) and static stretch. There was a significant increase in hamstring length regardless of treatment but no significant difference between treatments. Similarly, the comparison among PNF stretching alone (isometric contraction for 10 s, five s rest for four times), PNF stretching and cold (immersed in a cold-water bath (8°C) for 10 minutes before performing the same PNF protocol), and PNF and heat (immersed in a hot-water bath (44°C) for 10 minutes before performing the same PNF protocol) for five consecutive days did not lead to any difference in hip range of motion.[71] Knight et al.[66] studied the effects of four treatments (static stretching alone, warm-up prior to static stretching, superficial moist heat for 15 minutes prior to static stretching, and seven minutes of continuous ultrasound prior to static stretching) on plantar flexor flexibility over six consecutive weeks. The use of ultrasound for seven minutes prior to stretching was the most effective treatment for increasing ankle dorsiflexion range of motion. Ultrasound

may have provided a deeper heat at the muscular level[72] compared to the hot pack or hot bath that might only increase skin temperature.

Massage

Stretching, warm-up, and massage are often performed in sport practice as treatment to prevent muscle injury. Rodenburg et al.[73] reported that the combination of these treatments could reduce some negative effects of muscle soreness induced by eccentric exercise. The application of warm-up aimed to decrease viscosity of muscle tissues and stretching aimed to reduce passive tension. These two treatments were performed before eccentric exercise, and massage was performed after exercise with the aim of increasing blood flow and reducing waste products. The results, however, were not consistent as the maximal force, the flexion of elbow angle, and the creatine kinase level in blood were reduced, while other soreness parameters such as soreness sensation, extension elbow angle, and myoglobin in blood did not change. Therefore, the combination of these treatments did not reduce the severity of muscle soreness any more than any individual treatment. In other studies, warm-up was reported to be effective in reducing the severity of muscle soreness and functional loss,[74] massage was effective in reducing soreness sensation,[75-77] while stretching did not show any effect at all.[3, 78, 79]

THE EFFECTS OF STRETCHING ON PERFORMANCE

Stretching is expected to increase flexibility, and, consequently, enhance sport performance.[9] The effects of stretching on several performance parameters have been investigated including muscle strength, power, and endurance, as well as the efficiency of exercise such as running economy. However, recent research[2, 7, 16, 80, 81] still questions whether these interventions provide any benefit for performance (see Table 5).

Insert Table 5 about here.

Muscle strength, power, and endurance

The acute effects of static stretching, ballistic stretching, and PNF can reduce muscle strength as determined by maximum lifting capacity[5, 12, 16] and isometric contraction force.[2] Nelson et al.[4] reported that a decrease in muscle strength for slow velocities of movement after static stretching, and decreases in performance of functional high velocity movements, such as jumping, after static stretching.[1, 4, 6, 7] The negative acute effect of stretching on performance is probably explained by the change in neuromuscular transmission and/or biomechanical properties of muscle. Several studies of the effect of stretching on performance have demonstrated a reduction of performance associated with a decrease in neural activation (H-reflex amplitude).[2, 43, 44] Some stretching research has reported an increase in muscle compliance as investigated by range of motion,[18, 22, 31, 69, 70] active muscle stiffness,[1, 40] and passive stiffness.[25, 26]

A study by Fowles et al.[2] assessed strength performance after prolonged stretch (13 maximal stretches, two minutes and 15 seconds hold, and five seconds rest) by measuring force, EMG activity, and passive stiffness. Strength was lost maximally immediately after stretch (28%) and lasted more than one hour after stretch (9%). Interestingly, muscle activation and EMG activity was significantly depressed after stretching but was recovered by 15 minutes. Passive stiffness recovered quickly after stretching at 15 minutes but did not fully recover within one hour. The results imply that the impaired muscle activation was responsible for strength loss after prolonged stretching in the early phase while impaired contractile force was responsible for strength loss throughout the entire period. This implication is consistent with the results of several studies where the mechanism responsible for a decrease in performance after acute stretching is likely to be caused by neuromuscular

inhibition.[1, 80] Behm et al.[80] investigated the effects of static stretching (held for 45 seconds, and rest for 15 seconds for five times) on voluntary and evoked force, and EMG activity of quadriceps. Maximal voluntary and evoked contraction decreased similarly by 12%, and muscle activation and EMG activity decreased 2.8% and 20%, respectively. Similarly, Cornwell et al.[1] reported that static stretching of gastrosoleus (180 s) reduced jump height by 7.4% but active stiffness was reduced by only 2.8%. Other studies reported that static stretching reduced jumping performance (knee bend) by 3%.[6, 7] A reduction in jumping performance was consistent with a reduction of EMG activity,[7] but there were no changes in biomechanical variables (vertical velocity, knee angle, duration of concentric and eccentric phases).[6] The prolonged stretch in Fowles et al.'s[2] study (75 seconds for 13 times) might have increased muscle compliance more than in any other study as there was evidence that static stretching held for 90 seconds for five times could decrease muscle stiffness.[25, 26] The shorter duration could not change passive muscle properties[18, 39] therefore, the change in muscle compliance and muscle inactivation in Fowles et al.'s[2] study caused more strength loss (28%) than that reported by Behm et al.[80] (12%).

The detrimental effects of acute stretching exercise on muscular endurance was shown by Laur et al.[82] when the application of acute stretching reduced the maximal number of repetitions performed with a submaximal load, and also produced higher perceived exertion scores. Although the magnitude of reduction was small, it was statistically significant.

Interestingly, studies on the effects of long-term stretching reported a positive effect of stretching on performance.[40, 57, 81] Three weeks of flexibility training in both PNF and static stretching increased peak torque of hamstrings eccentrically (at 60°/s and 120°/s) and concentrically (at 120°/s only).[57] PNF training (contract-relax technique) for eight weeks increased maximum torque of knee flexors and extensors.[81] The increase in muscle strength

of knee flexors was significant at all velocities and might be due to the contraction phase of the PNF stretching technique showing the same effect as isometric muscle training. Therefore, knee flexors, which are used in normal activity less than knee extensors, showed more increase in muscle strength. More functionally, Wilson et al.[40] reported that eight weeks of static flexibility training increased rebound bench press performance by 5.4%, in accordance with a decrease in active muscle stiffness by 7.2%. The authors proposed that flexibility-induced performance enhancement might result from increased musculotendinous compliance facilitating the use of energy strain in stretch short-cycle activities. In contrast with the acute stretching, Hunter and Marshall[83] reported that the combination of static and PNF training for ten weeks did not result in a detrimental effect on countermovement jump and drop jump, but helped to increase knee joint range of motion.[83] Therefore, flexibility training of at least three weeks is beneficial to some performance factors as indicated by increased range of motion and muscle strength. Unfortunately, there is no research on the effects of flexibility training on neuromuscular activity.

There are no studies on the effects of acute stretching after prolonged flexibility training. It would be questionable whether the negative effects of acute stretching will attenuate the positive effects of flexibility training because the athletes, who commonly undertake flexibility training, also perform stretching before competition. There are also no studies on the effects of dynamic stretching on performance. As research[2, 7, 16, 80, 81] reported the detrimental effects on athletic performance of all stretching techniques (static, PNF, and ballistic), dynamic stretching might be a useful protocol to increase flexibility without decreasing athletic performance.

The efficiency of exercise

Flexibility is considered to play an important role in the efficiency of movement[32] by enabling the use of elastic potential energy in muscle.[9] The more compliant muscle-tendon unit needs more contractile force to transmit to the joint and, consequently, causes a greater delay in external force generation.[9] A stiffer muscle would provide a more efficient transmission of contractile force production,[9] but this contradicts the aim of stretching, which intends to increase muscle-tendon unit compliance.

Craib et al.[84] reported that less flexible runners have showed a reduced aerobic demand during running (better running economy). The positive and significant correlation between range of motion and the aerobic demand of running presented in only two movements (external hip rotation and dorsiflexion) accounted for 47% of the variance observed in running economy. Craib et al.'s[84] study was cross-sectional, did not control the training programme of the runners, and did not consider other factors that might have influenced running economy such as kinematic, anthropometric, physiological, and cellular variables. In contrast, the flexibility training of hip flexors (three weeks)[85] and lower leg muscles (quadriceps, hamstrings, and gastrosoleus) (ten weeks)[86] resulted in increased range of motion but had no effect on running economy. None of the published papers in this area reported the effects of flexibility on running time, stride length, stride frequency, or the perception of fatigue. The optimal level of flexibility (static and dynamic) for running performance needs to be researched.

THE EFFECTS OF STRETCHING ON INJURY PREVENTION

Rate of injury

Despite performance of stretching generally being recommended before exercise to reduce the risk of injury, a recent review of the effects of stretching on the incidence of injury indicated inconclusive results.[87] The inconclusive results might be due to exercise-related injury being a complex phenomenon with physiological, psychological, and environmental factors involved. The majority of research in this area has been retrospective and does not provide a clear relationship between flexibility and injury.[9, 88, 89]

In one prospective study, Van Mechelen et al.[90] provided a standardised program of stretching exercises to runners and assessed the number of injuries after 16 weeks. There was no reduction in injury incidence per 1000 hours of running between the experimental (standardised program of stretching exercise) and the control (no stretching information) groups. In a study of army recruits, [91, 92] a pre-exercise stretching programme of 11-12 weeks did not reduce the risk of exercise-related injury. Fitness and age,[92] and the early detection of symptoms of overuse injuries[90] were more important factors for injury rather than the stretching exercise. An increase in range of motion (or static flexibility) resulting from stretching may not be necessary for a majority of sports such as running and swimming which do not require extreme ranges of motion. Dynamic flexibility might be more important because it represents the resistance of the muscle-tendon unit during movement. However, there is no research on the relationship between dynamic flexibility and rate of injury.

Muscle damage

In contrast to the general belief that stretching helps reduce the risk of muscle damage from unaccustomed eccentric exercise, previous research reported that both prolonged static and ballistic stretching (60 s for two stretches for each muscle) induced muscle damage.[13]

Moreover, no research has reported any benefit from stretching on the severity of muscle damage or muscle soreness either before[3, 78, 79] or after[78] exercise. The effects of stretching before and after eccentric exercise have been reviewed recently.[93] The lack of evidence for the benefits of stretching may be due to previous research only investigating static stretching. If the decrease in passive muscle stiffness is the key to reducing the severity of muscle damage, static stretching in these studies[3, 78, 79] was not held long enough to induce a decrease in passive stiffness (most studies used stretches held less than 90 s). Interestingly, a study by McNair et al.[35] reported that passive dynamic stretching did reduce muscle stiffness, while a study by McHugh et al.[64] reported that passive stiffness was related to the severity of muscle damage measured by strength loss, pain, muscle tenderness, and creatine kinase activity. Therefore, any stretching technique that can reduce passive stiffness might help to reduce the severity of muscle damage. To date, there are no published papers reporting the effects of dynamic stretching on the severity of muscle damage. The effects of other stretching techniques such as ballistic and PNF on the severity of muscle damage have also not been determined.

SUMMARY AND CONCLUSIONS

Despite stretching commonly being performed before exercise to enhance performance and reduce the risk of injury, there is limited scientific data to support the suggested benefits of stretching. Static and ballistic stretching have been shown to have detrimental effects on muscle strength and functional performances such as jumping, and to have inconclusive effects on the incidence of injury, no effects on the severity of muscle damage. Even though research has indicated that stretching is an effective treatment to increase static flexibility (range of motion), the effects on dynamic flexibility (muscle stiffness) are inconclusive given

the variation of the length of hold and the number of repetitions used in studies. The aim of stretching is to increase flexibility, but does flexibility help to enhance performance? The ideal flexibility for the performance of each sports activity is different. Compliant muscle might be beneficial to eccentric contraction while stiffer muscle might be more suitable for concentric and isometric contractions. Does flexibility help to reduce the rate of injury? The majority of research does not support this statement. In fact, the majority of movement in sports requires repetitive movements within the normal range of motion. An increase in range of motion, therefore, is not necessary. The aim to reduce resistance during repetitive movement might be more beneficial in terms of increasing quality of movement and reducing the risk of overuse injury. Practically, the optimal level of flexibility is required because the increase in flexibility (more compliant muscle) might not benefit performance but may help to reduce the risk of injury. The compliant muscle-tendon unit absorbs and requires more energy to shorten, and consequently delays and reduces external force production. Nevertheless, the increase in ability to absorb energy in the compliant muscle might help to reduce the mechanical overload on muscle fibres, and consequently reduce the risk of muscle injury and the severity of muscle damage. Further research is needed to investigate the appropriate stretching techniques and the optimal level of flexibility which can maintain or improve performance, or which can prevent injury.

RECOMMENDATIONS

In order to clarify the effects of stretching, further research is recommended to:

- Provide information on the relationship of dynamic flexibility, performance, and rate of injury.

- Examine the effects of several stretching techniques such as ballistic, PNF, and dynamic stretching on dynamic flexibility and neuromuscular sensitivity.
- Compare the effects of several stretching techniques such as static, ballistic, PNF, and dynamic stretching on different types of performance, the severity of muscle soreness, running economy, and rate of injury.
- Study the effects of acute stretching after long-term flexibility training.
- Provide more information on the appropriate flexibility level to enhance performance and reduce the risk of injury.

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Table 1: Summary of the advantages and disadvantages of stretching techniques.

Techniques	**Definition**	**Advantages**	**Disadvantages**
Ballistic stretching.	Repetitive bouncing movements at the end of joint range of motion.[8]	Increased range of motion.[8]	Reduced muscle strength.[12] May cause injury.[13]
Proprioceptive neuromuscular facilitation (PNF) stretching.	Reflex activation and inhibition of agonist and antagonist muscles.[14]	Increased range of motion.[15]	Reduced jump height.[16] Need experience and practice.[17]
Static stretching.	Passive movement of a muscle to maximum range of motion and then holding it for an extended period.[8]	Increased range of motion.[18] Simple technique.	Reduced muscle strength.[1, 2] May cause injury.[13]
Dynamic stretching.	Slow movement of a joint as a result of antagonist muscle contraction throughout the range of movement.[8]	Unknown.	Unknown.

Table 2: The effects of static stretching on muscle properties.

References	Trial design	Sample	Interventions	Outcome measures	Main results
ROM and passive stiffness					
McHugh et al.[34]	PPT	9 men and 6 women (hamstrings).	Static stretch (hold 45s) 1. At onset of EMG. 2. Five degrees below the onset of EMG (negligible EMG activity).	1. Peak torque. 2. ROM. 3. EMG.	S: ↓ torque. ↑ ROM.
Magnusson et al.[33]	PPT	10 men (hamstrings).	1. Static stretch (hold 90s, rest 30s) 5 times (stretch 1-5). 2. Repeated static stretch one time (stretch 6).	1. Peak torque. 2. ROM. 3. EMG.	S: ↓ stress relaxation. ↑ ROM.
Magnusson et al.[25]	PPT	7 women (one leg stretch one leg control) (hamstrings).	Static stretch (45s hold x 15-30s rest x 5times), twice daily, 20 consecutive days.	1. Stress relaxation. 2. Energy. 3. EMG. 4. ROM.	S : ↑ ROM.
Halbertsma et al.[18]	RCT	10 men and 6 women with short hamstrings.	1.Static stretching (30 s hold x 30 s rest) for 10 min (n=10) 2.Control-rest (n=6)	1. Peak torque. 2. ROM. 3. Passive stiffness.	S: ↑ ROM.
Magnusson et al.[26]	CCT	8 neurological intact and 6 spinal cord injury volunteers (hamstring).	Static stretch (hold 90s).	1. Stress relaxation. 2. Passive torque. 3. EMG.	NS
Magnusson et al.[10]	PPT	13 men (hamstrings).	5 static stretches (hold 90s, rest 30s) and repeated 1 hr later.	1. Stiffness. 2. Energy. 3. Passive torque.	S: ↓ energy, stiffness, and peak torque.

Klinge et al.[29]	CCT	12 men in experimental group, 10 men in control group.	4 x 45 s static stretch.	1. ROM. 2. Passive stiffness.	NS
McHugh et al.[28]	CCT	8 men and 8 women (hamstrings).	SLR stretch.	1. Peak torque. 2. ROM. 3. EMG.	S: ↑ ROM.
Magnusson et al.[24]	CCT	12 men (hamstrings)	1. 90 s static stretches. 2. Continuous movements 10 times at 20°.s^{-1}.	1. ROM. 2. Passive stiffness.	S: ↑ ROM.
Muir et al.[23]	RCT	10 men (one leg-stretching, one leg-control) (hamstrings).	1. Static stretching (30sx10s) for 4 times. 2. Control-rest.	1.Peak torque 2. Centre range (of hysteric loop).	NS
McNair et al.[35]	CBT	15 men and 8 women (plantar flexors).	Static stretching 1. 1x60s hold. 2. 2x30s hold. 3. 4x15s hold. 4. Continuous passive movement for 60s.	1. Passive stiffness. 2. Peak torque.	*Continuous movement* S: ↓ passive stiffness. *Hold condition* S: ↓ peak tension.
Magnusson et al.[39]	PPT	20 men	3 static stretches (hold 45s, rest 30s) and repeated 1 hr later.	1. Stiffness. 2. Energy. 3. Passive torque.	S: ↓ stress relaxation.
Kubo et al.[36]		7 men (plantarflexors).	Passive stretching to 35° dorsiflexion at 5°.s^{-1} for 10 min.	1. Tendon stiffness. 2. Tendon hysteresis. 3. MVC.	S: ↓ tendon stiffness (10%), tendon hysteresis (34%).

Kubo et al.[38]	CBT	8 men (plantarflexors).	Passive stretching to 35° dorsiflexion at 5°.s^{-1} for 5 min.	1. Tendon stiffness. 2. Tendon hysteresis.	S: ↓ tendon stiffness (8%), tendon hysteresis (29%).
ROM and active stiffness					
Wilson et al.[40]	CCT	16 male weightlifters (n = 9 in experiment, n = 7 in control group).	Flexibility training (6-9 rep) of upper extremities, 10-15 min per session, twice a week for 8 weeks.	1.Rebound bench press (RBP). 2.Purely concentric bench press (PCBP).	S: ↑ ROM (13%). S: ↑ RBP (5.4%). S: ↓ SEC stiffness (7.2%).
McNair and Stanley[31]	CCT	12 men and 12 women (plantarflexors).	1. Static stretch (30sx30s). 2. Jogging (60%MHR). 3. Combined 2+1. Randomly order, each intervention for 10 min.	1. ROM. 2. Active stiffness.	*Jogging group* S: ↓ active stiffness. *All groups* S: ↑ ROM.
Cornwell et al.[30]	PPT	10 men (plantarflexors).	Passive stretching.	1. ROM. 2. Active stiffness.	S : ↑ ROM.
Hunter and Marshall[55]	CCT	15 men, 15 women (n =15 in experiment and control groups) (plantarflexors).	10 x 30 s static stretches.	Active stiffness.	NS

Cornwell et al.[1]	PPT	10 men (plantarflexors).	Passive stretching (30 s x 6 times).	1. Active muscle stiffness. 2. EMG. 3. Jump height.	S: ↓ jump height (7.4%). ↓ active stiffness (2.8%).

CCT = controlled clinical trial; RCT = randomised controlled trial; PPT = pre- & post-test trial; CBT, counterbalance trial; ROM = range of motion; EMG = electromyography; SEC = series elastic components; S = significant; NS = non-significant.

Table 3: The effects of static stretching on neuromuscular activity.

Reference	Trial design	Samples	Interventions	Outcome measures	Main results
Thigpen et al.[48]	CCT	6 men, 2 women.	3 x 20 s toe touch stretching.	H-reflex.	S: ↓ H/M ratio 21.49%.
Vujnovich and Dawson[43]	CCT	*Group A* (n=14): static stretching. *Group B* (n=2): Similar as A but followed up every 2 min for 10 min. *Group C* (n=5): Similar to B but followed by ballistic stretch (1 rad.s^{-1} for 160 s). *Group D* (n=2): Stretching at midway between neutral and fully	Maximally dorsiflexion for 160 s.	H-reflex.	*Group A*: S: ↓ H-reflex (45%). *Group B*: S: ↓ H-reflex during stretching but NS afterward. *Group C*: S: ↓ H-reflex during ballistic stretching (84%) >static stretching (40%). *Group D*: S: ↓ H-reflex 40%.

		dorsiflexion.			
Rosenbaum et al.[45]	CCT	50 male athletes.	Three min static stretch: hold 30s.	H-reflex of triceps surae: 1. Peak force. 2. Force rise rate. 3. Half relaxation rate. 4. EMG amplitude & integral. 5. EMG latencies. 6. Impulses.	S: ↓ peak force, force rise rate, half relaxation rate, EMG amplitude and integral. S: ↑ EMG latencies.
Avela et al.[44]	CCT	6 men (plantar flexors).	Repeated passive stretching (1 hr).	1. MVC. 2. 50%MVC. 4. H_{max} 5. Motor unit firing rate (ZCR).	S: ↓ MVC (23.2±19.7%). S: ↓ H reflex (46.1±38.3%). S: ↓ ZCR (12.2±11.4%).
Guissard et al.[53]	PPT	7 men, 4 women.	Static stretch at 10° and 20°.	H/M ratio.	*Dorsiflexion (10°)* S: ↓ H/M ratio (25%). *Dorsiflexion (20°)* S: ↓ H/M ratio (55%).

CCT = controlled clinical trial; RCT = randomised controlled trial; PPT = pre- & post-test trial; CBT, counterbalance trial; ROM = range of motion; EMG = electromyography; SEC = series elastic components; S = significant; NS = non-significant; MVC = maximum voluntary contraction; H/M ratio = H-reflex/M-wave ratio.

Table 4: The effects of PNF stretching on biomechanics and neuromuscular activity.

Reference	Trial design	Samples	Interventions	Outcome measures	Main results
Sady et al.[60]	CCT	1. Control (n = 10). 2. Static (n = 10). 3. Ballistic (n = 11). 4. PNF (n = 12).	1. Static: 3 x 6 s. 2. Ballistic: repeated movements for 20 times. 3. PNF: 3 x 6 s. 3 days/week for 6 weeks.	ROM.	PNF and control group. S: ↑ ROM.
Toft et al.[61]	PPT	10 men	Contract-relax (8 s maximum contraction, 2 s relax, 8 s static stretch) 6 times.	Stress relaxation.	NS
Magnusson et al.[25]	CCT	7 women (one leg stretch one leg control) (hamstrings).	Static stretch (45s hold x 15-30s rest x 5times), twice daily, 20 consecutive days.	1. Stress relaxation. 2. Energy. 3. EMG. 4. ROM.	S: ↑ ROM.
Magnusson et al.[26]	CCT	8 neurological intact and 6 spinal cord injury volunteers (hamstring).	Static stretch (hold 90s).	1.Stress relaxation. 2.Passive torque. 3.EMG.	NS

CCT = controlled clinical trial; RCT; PPT = pre- & post-test trial; ROM = range of motion; EMG = electromyography; SEC = series elastic components; S = significant; NS = non-significant.

Table 5: The effects of stretching on performance.

References	Trial design	Samples	Interventions	Outcome measures	Main results
Static stretching					
Kokkonen et al.[5]	CBT	15 men & 15 women (hamstrings).	20 min stretching (5 stretches, 3 tims assisted, 3 times unassisted, hold 15 s, rest 15 s).	1. Sit & reach score. 2. Maximum strength (1RM).	S: ↑ ROM (16%). S: ↓ strength (7.3%).
Fowles et al.[2]	CBT	8 men, 4 women.	13 x 135 s static sttretches, total 30 min.	1.MVC. 2.Twicth interpolation with EMG. 3. Twich characteristics at pre, immediately post, 5, 15, 30, 45, 60 min post-stretching.	S: ↓ MVC (28, 21,13,12,10, and 9% (by the time to collect data)). S: ↓ Motor unit activation & EMG after treatment but recovered by 15 min.
Knudson et al.[6]	CBT	10 men & 10 women (quadriceps, hamstrings, plantarflexors).	3 x 15 s static stretch.	1. Peak velocities. 2. Duration of concentric phase. 3. Duration of eccentric phase. 4. Smallest knee angle. 5. Jump height.	NS
Nelson et al.[4]	Pre-Post	10 men and 5 women	One active and 3 passive stretching for 15 min.	Peak torque at 1.05, 1.57, 2.62, 3.67, and 4.71 $rad.s^{-1}$.	S: ↓ strength at 1.05 $rad.s^{-1}$ (7.2%) and 1.57 $rad.s^{-1}$ (4.5%).
Behm et al.[80]	CBT	12 men	Quadriceps stretching (45s held,	1. MVC. 2. EMG.	S: ↓ MVC (12%), muscle

			15 s rest, for 5 sets).	3. Evoked torque. 4. Tetanic torque.	inactivation (2.8%), EMG (20%), evoked force (11.7%).
Cornwell et al.[1]	CCT	10 men.	3 x 30 s static stretch.	1.Active muscle stiffness. 2.EMG. 3.Jump height.	S: ↓ jump height (7.4%). S: ↓ active stiffness (2.8%).
Young et al.[7]	CBT	13 men, 4 women (quadriceps and plantarflexors).	2 x 30 s for each muscle.	1. Concentric force. 2. Concentric jump heigth. 3. Concentraic rate of force developed. 4. Drop jump height.	S: ↓ concentric force (4%).
Laur et al.[82]	CBT	16 men and 16 women (hamstrings).	3 x 20 s static stretching.	Perceived exertion.	S: ↑ perceived exertion.
Ballistic stretching					
Nelson et al.[12]	CBT	11 male and 11 female college students.	Ballistic stretch: 15 bob up and down once per min.	1. Sit & reach score. 2. Maximum strength (1RM).	S: ↑ ROM (7.5%). S: ↓ strength (7.3%).
PNF stretching (short term)					
Wiktorsson-Moller et al.[22]	CBT	8 healthy males.	PNF: isometric contraction 4-6 s, relax 2 s, passive stretching 8 s.	1. ROMs of lower extremities. 2.hamstrings and quadriceps strength.	S:↑ ROM of ankle dorsiflexion and plantarflexion, hip flexion, extension,

					abduction, knee flexion.
Church et al.[16]	CBT	40 women.	1. Static stretching. 2. PNF.	1.Vertical jump. 2. ROM.	PNF S: ↓ jump height (3%).
PNF stretching (long term)					
Wilson et al.[40]	CCT	16 male weightlifters (n = 9 in experiment group, n = 7 in control group).	Flexibility training (6-9 rep) of upper extremities, 10-15 min per session, twice a week for 8 weeks.	1.Rebound bench press (RBP). 2.Purely concentric bench press (PCBP).	S: ↑ ROM (3%). S: ↑ RBP (5.4%). S: ↓ SEC stiffness (7.2%).
Worrell et al.[57]	CCT	19 participants with short hamstrings (one leg static – one leg PNF)	Static-15 s held, 15 s rest PNF-5 s isometric, 5 s rest 4 rep per day, 5 days/week, 3 weeks	1. ROM 2. Con/ecc strength	S: ↑ strength Ecc-60 & 120°.s^{-1}. Con- 120°.s^{-1}.
Hande et al.[81]	CCT	16 men (one leg stretch, one leg control).	CR for 8 weeks (isometric at 70%MVC, 1-2 s rest, 10-15 s passive stretching). Follow up at 0, 4, 8 weeks.	Knee flexion & extension Con: 240, 180, 120, 60°.s^{-1} Ecc: 60 & 120°.s^{-1}.	S: ↑ torque *Extension*: ecc at 120 & 60°.s^{-1}. *Flexion*: all velocities.
Hunter and Marshall[83]		60 participants (15 per group).	Static tretching (3 x 20 s) and PNF (submaximal contraction 10 s).	1.Drop jump. 2.Countermove-ment jump.	NS

CCT = controlled clinical trial; RCT = randomised controlled trial; PPT = pre- & post-test trial; CBT, counterbalance trial; ROM = range of motion; EMG = electromyography; SEC = series elastic components; S = significant; NS = non-significant; MVC = maximum voluntary contraction; con = concentric contraction; ecc = eccentric contraction; RM = repetitive maximum.