

Mechanisms Underlying Performance Impairments Following Prolonged Static Stretching Without a Comprehensive Warm-up

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Abstract

Whereas a variety of pre-exercise activities have been incorporated as part of a “warm-up” prior to work, combat, and athletic activities for millennia, the inclusion of static stretching (SS) within a warm-up has lost favour in the last 25 years. Research emphasised the possibility of SS-induced impairments in subsequent performance following prolonged stretching without proper dynamic warm-up activities. Proposed mechanisms underlying stretch-induced deficits include both neural (i.e. decreased voluntary activation, persistent inward current effects on motoneurone excitability) and morphological (i.e. changes in the force-length relationship, decreased Ca^{2+} sensitivity, alterations in parallel elastic component) factors. Psychological influences such as a mental energy deficit and nocebo effects could also adversely affect performance. However, significant practical limitations exist within published studies, e.g. long stretching durations, stretching exercises with little task specificity, lack of warm-up before/after stretching, testing performed immediately after stretch completion, and risk of investigator and participant bias. Recent research indicates that appropriate durations of static stretching performed within a full warm-up (i.e. aerobic activities before and task-specific dynamic stretching and intense physical activities after SS) have trivial effects on subsequent performance with some evidence of improved force output at longer muscle lengths. For conditions in which muscular force production is compromised by stretching, knowledge of the underlying mechanisms would aid development of mitigation strategies. However, these mechanisms are yet to be perfectly defined. More information is needed to better understand both the warm-up components and mechanisms that contribute to performance enhancements or impairments when SS is incorporated within a pre-activity warm-up.

Key words: flexibility; range of motion; warm-up; sport; muscle morphology; neural.

Abbreviations

ECM	extracellular matrix
EMD	electromechanical delay
EMG	electromyography
E-reflex	exteroceptive reflex
GABA	gamma aminobutyric acid
GTO	Golgi tendon organ
H-reflex	Hoffman reflex
MEP	motor evoked potential
MTU	muscle tendon unit
MVC	maximal voluntary contraction
MVIC	maximal voluntary isometric contraction
NMES	neuromuscular electrical stimulation
PEC	parallel elastic component
PF	plantar flexors
PIC	persistent inward current
RNS	reactive nitrogen species
ROM	range of motion
ROS	reactive oxygen species
SS	static stretching
TMS	transcranial magnetic stimulation
TVR	tonic vibration reflex

Introduction

Pre-activity preparations for work, combat, and sports have been evident for millennia. It is surmised that pre-activity warm-ups would have been essential for successful movement execution in boxing, martial arts, and wrestling, which have been practiced in eastern civilizations such as the Chinese, Japanese, Aleut and Mongolians since pre-historic times (Draeger 1969) because of the extreme positions, kicks and strikes used in these activities. Stretching was also prescribed as part of exercise to prevent illness by Hua Tuo, the Han Dynasty physician (104-208 CE) (Kunitz 2016). In western civilizations, ancient Egyptians and Greeks held athletic competitions (Kunitz 2016) that would also have necessitated some form of warm-up to ensure success and prevent injury (Behm 2018). Jaquet et al. (2015) highlighted that the thrusting actions of medieval armoured fighters required a great range of motion for the

adduction/abduction of the shoulder and flexion/extension of the elbow. Hence, it is speculated that pre-exercise dynamic warm-up activities would have been predominant throughout history (Kunitz 2016), evolving into the mid-19th century as activities for recreation and health (e.g. Swedish Ling's gymnastics) and further morphing into dynamic and ballistic fitness and conditioning movements of the 1950s (e.g. 5BX exercise program for military personnel) (Orban 1962). Subsequently, during the 1960s and for approximately the next 30-40 years, static stretching (SS) replaced ballistic and dynamic stretching as a predominant warm-up activity, in addition to purposeful task-specific warm-ups. The goal of SS was to increase range of motion (ROM), decrease injury incidence, and improve athletic performance (Young 2007; Young and Behm 2003).

However, research around the new millennium started to accumulate showing that SS might impair rather than enhance athletic performance (Behm et al. 2001; 2004; Fowles et al. 2000; Kokkonen et al. 1998; Power et al. 2004). As the reports of SS-induced performance deficits multiplied, professionals and practitioners increasingly replaced SS with dynamic stretching as the primary flexibility component of the warm-up (Judge et al. 2020). However, in the last decade several critical reviews have disseminated the idea that numerous limitations in study design within the SS literature may have biased the research findings underlying the shift away from SS. A number of reviews (Behm et al. 2016a; Behm and Chaouachi 2011; Chaabene et al. 2019; Kay and Blazevich 2012; Lima 2019) highlighted that prolonged periods of acute SS (e.g. > 60 s per muscle group) generally induced significant and practically relevant deficits while shorter SS had trivial effects. In addition to excessive SS durations, there were also concerns relating to ecological validity in many studies. Behm et al. (2016a) documented that many SS studies did not employ a prior aerobic-based warm-up, did not include dynamic sport-

specific activities after stretching, conducted the testing within 3-4 min of the experimental protocol (when longer periods are common in many sporting situations), and were influenced by possible placebo effects associated with participant bias (i.e. sport science students who were taught or had read that SS impairs performance) (Bertolaccini et al. 2019; Blazevich et al. 2018; Janes et al. 2016). A number of recent studies have shown that when short- or moderate-duration (<60 s per muscle) SS is employed within a comprehensive task- or sport-specific warm-up, the effects on performance are typically trivial or even positive (Blazevich et al. 2018; Mascarin et al. 2015; Murphy et al. 2010; Reid et al. 2018; Samson et al. 2012).

Although both recent reviews and original investigations utilising appropriate durations of SS within a full warm-up have revealed significantly increased ROM with trivial or positive effects on performance, it is still commonly reported that an acute bout of SS impairs subsequent performance. A recent survey of 195 American soccer coaches reported that 134 coaches only used dynamic stretching, 35 used a combination of dynamic and SS, one coach used only SS and others used a variety of ballistic, proprioceptive neuromuscular facilitation and other techniques (i.e. foam rolling) during warm-up (Judge et al. 2020). The change in public perception and athletic practice is problematic as this change is likely a consequence of recommendations from research with limited ecological validity for athletic practice.

In contrast, prolonged SS is still prescribed for individuals with serious range of motion limitations, especially in rehabilitation and other clinical settings (Decoster et al. 2005). So there is still a need to develop interventions to reduce or mitigate the effects of prolonged SS when employed in isolation (i.e. without further warm-up or task practice). This would require a complete understanding of the mechanisms by which stretching affects force production, however, these are yet to be fully described. Thus, the objective of this narrative review is to

highlight the existing and most recent literature on the effects of SS on subsequent performance with the major focus to examine the neurological, morphological, and psychological mechanisms underlying SS-induced performance alterations.

Effect of Static Stretching (SS) on Physical Performance

The position that static stretching could enhance muscle force production was borne out by the results of the limited research up to the 1990s. For example, Worrell et al. (1994) implemented four hamstrings stretches of 15-20 s each (60-80 s) and found improvements in leg flexion concentric and eccentric contraction torques of 8.5% - 13.5% and 2.5% - 11.2%, respectively. However, results of research in the mid- to late 1990s led to the questioning of the legitimacy of the belief that SS could improve performance. Kokkonen et al. (1998) was one of the first studies to report SS-induced performance impairments when they found that 5 stretches performed for 6 repetitions of 15 s (90-s total per stretch) decreased knee flexion and extension torque by 7-8%. A subsequent paper by Fowles et al. (2000) then triggered an avalanche of further research. They imposed 13 static plantar flexor (PF) stretches of 135 s each (~30 min total stretching) and observed a 28% mean decrease in PF maximal voluntary isometric contraction (MVIC) force immediately post-stretch with a 9% deficit still observed after 60 min. Behm et al. (2001) imposed a briefer, but still very long, 20-min SS on the quadriceps and found impairment in MVIC (12%), electromyographic (EMG) activity (20%) and evoked twitch force (12%) after stretching. However, many researchers have investigated the impact of much shorter periods of SS. A review by Behm and Chaouachi (2011), recommended that stretching durations of <90 s would tend to produce trivial magnitude performance impairments. Later, Kay and Blazeovich (2012) and Behm et al. (2016a) concluded that stretches <60 s would produce trivial

performance impairments, whereas the risk of longer periods of stretch was significantly greater. Behm et al. (2016a) found that strength impairments in studies incorporating <60 s of SS per muscle were 2.8% compared to a 5.1% reduction when using ≥ 60 s of SS. With SS studies incorporating power- or speed-based variables, mean power impairments averaged 2.6% with ≥ 60 s of SS. Studies that used <60 s of SS showed trivial power or speed performance deficits, averaging only 0.15%. Since these reviews were published, numerous studies have directly compared the effects of shorter (e.g. <60 s) versus longer (≥ 60 s) periods of stretch on physical performance outcomes and generally found that impairments existed when stretches lasted ≥ 60 s (Palmer et al. 2019; Reid et al. 2018; Vieira 2019). Notwithstanding this general finding, some research has suggested that the cumulative duration of SS may also impact subsequent performance. For example, Brusco et al. (2018) reported countermovement jump height deficits following 30 s SS of multiple muscles (calves, hamstrings, gluteus maximus and quadriceps). Thus, several recent reviews (2011 – 2016) as well as intervention studies highlighted that performance impairments were more commonly associated with >60 s of SS, although some cumulative effect might be conferred when multiple muscles are stretched for a reasonable (as yet undefined) time. Despite this, it can be questioned whether publishing sports scientists took notice, or governing bodies altered/reversed position stands or exercise prescription recommendations that supported the removal of static stretch during a warm-up. **One benefit of studying longer durations of stretch, which tend to produce significant losses in muscle function, is that it more easily allows for the study of the mechanisms underpinning the loss. However, it should be clarified that these studies have practical implications for only a small number of individuals (who perform long durations of stretch with little/no other warm-up and then produce maximal-effort tasks). Both authors and readers should thus be clear that the results of studies**

using these designs may be misinterpreted or mistakenly applied in athletic, rehabilitation or fitness circumstances.

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Search Strategy

A literature search was performed by the co-authors using PubMed, SPORTDiscus, Web of Science, and Google Scholar databases. The topic was systematically searched using a Boolean search strategy with the operator “AND” and keywords related to stretching, flexibility, range of motion, AND measures of performance (i.e. strength, power, endurance). Based upon our knowledge of the area we also contributed additional studies which we had knowledge of but were not picked up in systematic searches; further, we conducted searches of our personal computer databases for related articles.

Search Results

Table 1 reports a sample of 44 studies published since the Behm et al. (2016a) review that examined the effect of SS on performance. The table highlights that some of the methodological difficulties presented above are still prevalent in the literature. First, as with many areas of sports science research, female participants were under-represented (25.5% of the total sample). Also, only two of 27 studies investigated youth (<18 years of age) and no studies examined participants over 40 years of age. Therefore, potential effects of sex and age on the stretch-induced loss of muscle force cannot be clearly described as yet. Second, several issues relating to study validity were highlighted by Behm et al. (2016a). One issue was the importance of a complete warm-up involving dynamic muscular activities both before and after SS. However, prior aerobic or dynamic warm-up activities were included in only 50% of the studies published since 2016, and only four studies (11.3%) included dynamic activities post-SS. The

four SS studies in which a full warm-up was included and stretches were held for <60 s per muscle group reported either trivial effects on performance (Blazevich et al. 2018; Reid et al. 2018) or small performance improvements (Murphy et al. 2010; Samson et al. 2012). In these four studies, multiple muscle groups were stretched resulting in an accumulation of more than 60 s of total stretching, yet with comprehensive warm-ups there was still no evidence of performance impairments. The majority of studies (59.1%) imposed tests less than 5 min post-stretching, even though longer periods usually separate athletic activities (i.e. training session, competition) from SS, during which time further sport-specific dynamic activities are completed and athletes return to dressing room for equipment adjustments, pre-game coach's strategy and motivational talks, and other pre-match activities (Behm et al. 2016a). Regardless, the overall results of these 27 studies reveal a mean moderate magnitude (8.04%; $d=0.55$) ROM increase and a mean small magnitude (-1.5%; $d=0.36$) post-SS performance decrement. Hence, even though research protocol limitations persist, which likely amplify the stretch-induced performance loss but also limit ecological validity under many conditions, SS-induced performance decrements were observed to be small on average in studies after 2016.

Based on the above arguments, it appears important to consider that stretch-induced performance decrements are likely to be negligible when SS is of short or moderate duration (e.g. <60 s per muscle), at least when only a few muscles are stretched and/or a complete physical preparation (warm-up) is performed between the SS and exercise or sporting task. It is also notable that, in contrast to the commonly reported performance impairments, five studies that reported strength decrements at short muscle lengths (-10.2%) observed moderate strength improvements at the longest muscle lengths tested (+2.2%) (Balle et al. 2015; Herda et al. 2008; McHugh and Nesse 2008; McHugh et al. 2013; Nelson et al. 2001b). Performance enhancement

at longer muscle lengths could be of practical importance since muscle strain injuries are more likely to occur with the muscle at a longer rather than shorter length (Behm et al. 2016a; Heiderscheit et al. 2010) and many sporting activities require force production at longer muscle lengths. Furthermore, SS-induced performance enhancement has also been reported during stretch-shortening activities that involve prolonged transition (eccentric to concentric) phases such as running at slower velocities (Godges et al. 1989) and the rebound bench press (Wilson et al. 1992).

Regardless of the background of evidence that properly-programmed acute SS may not be associated with performance decrement, or may moderately enhance performance, longer durations of SS are needed by some individuals in some sports, or in some clinical and rehabilitation settings. In these cases, there remains a possibility of stretch-induced performance decrement that could impact functional performance and clinical/rehabilitation outcomes, both acutely and in the longer term. **When longer durations of SS are necessary, athletes, coaches and clinicians may have to consider the potential trade-offs or concessions between substantial increases in flexibility and performance decrements. Future applied stretch research should investigate interventions that would allow more prolonged SS without inducing impairments (i.e. alternative warm-up strategies, timing, nutritional and pharmaceutical aids).** In order to develop these interventions that might minimise the effects of SS on muscle functional loss it is first important to determine the mechanisms that underpin the phenomenon. Secondly, it is of additional interest to determine which mechanisms are involved to counterbalance performance impairments when a full warm-up is incorporated.

Neural Responses to Static Stretching (SS)

Changes in Muscle Activity (Electromyography: EMG)

The EMG signal **broadly reflects the extent of muscle activation** and is a composite signal that is influenced by both central and peripheral components of the neuromuscular system including muscle conduction velocity, motor unit recruitment and firing frequency (rate coding), motor unit synchronisation, and muscle fibre action potential amplitudes and durations (Farina et al. 2002; Hagg 1992). Stretching can influence sensory (afferent) inputs, modulating both supraspinal (**brain**) and spinal excitability (Matthews 1981). However, the effect of SS on muscle activation capacity as measured by EMG activity is often contradictory throughout the SS literature. EMG decrements have been reported after a variety of SS durations when tested either immediately (<1 min) (Babault et al. 2010; Behm et al. 2019; Marchetti 2017; Ryan et al. 2014; Trajano et al. 2013) or 10 (Behm et al. 2001), 15 (Fowles et al. 2000) or 60 min (Avela et al. 1999) post-SS. In contrast, there were no significant EMG reductions after SS protocol durations of 30-120 s (Caldwell et al. 2019; Palmer et al. 2019; Power et al. 2004; Reid et al. 2018), 180 s (Kay and Blazevich 2009a), or 5 (Mizuno et al. 2014), 9.2 (Herda et al. 2008) or 10 min (Barbosa et al. 2019). Mixed results were also reported by Damasceno et al. (2014) who recorded both increased biceps femoris EMG activity and no change in the vastus medialis or gastrocnemius EMG during a 3-km running time trial following 3×30 s of SS. In a series of studies, Trajano et al. (2013; 2014a; 2019) monitored EMG immediately, 15 and 30 min post-SS and reported impairments immediately post-SS but not at the latter testing points, suggesting an initial impairment but reasonably rapid recovery of muscle activation. In addition to M wave-normalised EMG amplitudes, Trajano et al. (2013) measured the V-wave amplitude (variant of the H-reflex providing evidence of voluntary drive to the motoneurons) and found that both EMG and V-wave changes were correlated with the decrease and subsequent recovery of MVC

force. Correlations between SS-induced reductions in EMG amplitude and MVC force have been also reported in other studies (Fowles et al. 2000; Kay and Blazevich 2009a; Trajano et al. 2017).

While SS-induced influences upon the EMG signal may be relatively transient, the muscle action potential wave (M-wave) amplitude and duration (reflects changes in electrode recording volume) can be affected by more persistent peripheral changes such as exercise-induced changes in intramuscular and interstitial acidity, electrolyte balance (i.e. Na^+ , K^+ , Ca^{2+}), contraction-induced ischaemia, electrode recording volume, and other factors (Dimitrova and Dimitrov 2003). SS elicits vasoconstriction reducing blood flow to the stretched muscle (Venturelli 2019) due to a systemic increase in sympathetic neural tone (Cui et al, 2006) from the stress induced by SS on the mechano- and metaboreceptors (Venturelli 2017). As this SS-induced vasoconstriction is followed by a hyperaemic response, the muscles supply of oxygen, substrates, and removal of CO_2 and metabolites can vary substantially, modulating energy dependent processes such as the muscle action potential's $\text{Na}^{++}/\text{K}^+$ pump. Furthermore, M-wave amplitudes change when joints are passively rotated, possibly because of changes in muscle architecture changing the relative position of the electrodes relative to the muscle fibre (Frigon et al. 2007; Vieira et al. 2017). Therefore, small changes in muscle length (or width) caused by stretching could change both M-waves and EMG amplitudes. Also, changes in recording conditions (e.g. changes in electrode-skin impedance with sweat for example, electrode movement) could also affect the recordings. Hence in addition to the possibility of electrode displacement with stretching, it is important to normalize the EMG signal to the present circumstances of the M-wave. Thus, evidence of extensive, full motor unit depolarisation (activation) throughout the muscle may be obscured by an EMG signal that is depressed by peripheral changes, as can be observed by measurement of the M wave. Secondly, the EMG-

force relationship is more commonly described by a curvilinear slope (Perry and Bekey 1981; Solomonow et al. 1990) and thus incremental changes in EMG do not translate directly into similar changes in muscle force or power. Muscle activation measures, including EMG activity (Perry and Bekey 1981; Solomonow et al. 1990) and the interpolated twitch technique (in which stimulation is evoked during and after a voluntary contraction to determine the extent of muscle inactivation)(Behm and St-Pierre 1997) tend to plateau at maximal and near maximal muscle contraction levels, suppressing evidence of stretch-induced neural deficits with strength or power testing. Thus, this muscle activation-force plateau may not allow small changes in activation to be detected during near-maximal muscle exertions. The conflicting EMG changes among the many SS studies due to the aforementioned variables (i.e. EMG-force curvilinear relationship, peripheral effects on the M wave characteristics) prevents a conclusive statement relating to the effect of SS on EMG, and suggests the possibility that surface EMG recording might lack the sensitivity to be a strong and consistent proxy for central (neural) drive measurement. However, when muscle activation signals were normalized to the M wave or the MVC, there is more consistent observations of acute reductions in EMG (Blazevich et al. 2012a; Pulverenti et al. 2019; 2020), Hoffman (H-) reflexes (Budini et al. 2018b; Guissard et al. 2001) as well as stretch-training induced decreases in normalized H-reflexes (Blazevich et al. 2012b).

Reflex-induced EMG activity may also influence ROM. Guissard and Duchateau (2006) suggested that the maximal passive joint ROM is strongly associated with the degree of muscle resistance induced by tonic reflexes. Whereas dynamic movements and dynamic stretching tend to increase facilitatory reflex responses (Behm 2018), SS is purported to reduce facilitatory reflex (excitatory) activity (Behm et al. 2001; 2004; 2016a; Behm and Chaouachi 2011). While this reduced reflex activity may help enhance muscle relaxation, and thus speculatively

contribute to enhanced ROM, it might alternatively be expected to negatively impact the ability to activate the motoneurons and thus inhibit maximal activation of the stretched muscles. However, reflex effects are suggested to be more transient (Guissard et al. 1988), lasting only until the stretch is released or for seconds after, and thus probably contribute less significantly to performance impairments than changes in other neuromuscular properties.

Motoneurone Excitability (Hoffman (H) reflex amplitude)

As we have emphasised, while long durations of SS can negatively impact performance, the reflex inhibition may be brief. The H-reflex is a proxy measure of the afferent excitability of the spinal motoneurone (Schieppati et al. 1986; Zehr 2002). **It attempts to mimic a stretch reflex with the stimulation of a peripheral sensory nerve, and thus reflects the excitation or inhibition of the reflex circuit affecting the ability to produce force.** For example, Avela et al. (1999) reported that 1 hour of passive triceps surae SS reduced MVC force (23.2%), EMG (19.9%), and H-reflex activity (43.8%) immediately following SS, but no significant effects were observed at 15 and 30 min post-SS. Guissard et al. (1988) reported that SS-induced H-reflex deficits recovered quickly and were primarily only limited to the duration of the stretch. When implementing 30 s of SS, Budini et al. (2018a) reported no significant H-reflex impairments.

In the few studies in which H-reflex amplitudes were found to be impaired by SS, the cause was speculated to be due to a reduction in excitatory drive from the Ia afferents due to decreased resting muscle spindle discharge (disfacilitation) resulting from an increase in compliance within the muscle-tendon unit (Avela et al. 1999). Guissard et al. (2001) compared the activity of H-reflexes and exteroceptive (E-) reflexes **(receptors primarily located in cutaneous/ skin tissue)** during stretching and were able to delineate that the H-reflex (pre-

synaptic mechanisms) was attenuated with small stretching amplitudes whereas E-reflex (post-synaptic mechanisms) inhibition was predominant during large amplitude stretches. Pre-synaptic inhibition involves the release of inhibitory neurotransmitters that inhibit Ca^{2+} channels, reducing glutamate (excitatory neurotransmitter) release from nearby synapses (Stein 1995), whereas post-synaptic inhibition involves the release of neurotransmitters from the post-synaptic neurone that alters membrane conductance or membrane potentials (Stein 1995). Nonetheless, immediately following the cessation of stretching the H- and E-reflexes returned to their baseline values (Guissard et al. 2001). In contrast to their study in which stretch-induced H-reflex inhibition was not detected, another Budini et al. (2018b) study reported two stages of H-reflex inhibition with primarily post-activation depression of Ia afferents caused by maximal passive ankle dorsiflexion stretching within the first 18 s, then from 21-30 s there was a weaker post-activation depression, with inhibition from type II afferents or post-synaptic inhibition. However, there is evidence that H-reflex decrements may not be directly associated with performance changes. Stevanovic et al. (2019) found that both the H/M ratio (-20.5%) and vertical jump height (-2.6%) decreased 8 min following SS (6-min stretch duration), however when basketball-specific exercises were completed post-SS, vertical jump height was significantly increased 3.0% above baseline even though the H/M ratio decreased further (-30.2%). The relationship between H-reflex inhibition and performance impairments are somewhat conflicting. Although Stevanovic (2019) reported 8 min of H-reflex inhibition after stretching, most other studies report H-reflex inhibition during the stretch with a rapid return to resting values after stretching (Avela et al. 1999; Guissard et al. 1988; 2001). However, these post-stretch H-reflex measurements were taken with the muscle at rest (relaxed) and may not reflect changes that might occur during muscle contraction. In summary, based on the few available studies, SS-induced H-reflex depression may dissipate soon

after stretching and thus is unlikely to considerably negatively impact subsequent performance. Nonetheless, muscle spindle discharge is not the only reflex mechanism that may affect subsequent performance.

Exteroceptive (E) Reflexes

Myofascia and skin are densely innervated by sensory receptors (Schleip 2003a; 2003b) that are sensitive to skin stimulation, tangential forces and lateral stretch (Kruger 1987). E-reflexes are initiated by cutaneous (skin) receptors that have polysynaptic innervations to motoneurons (Jenner and Stephens 1982; Kearney and Chan 1999), which contribute to sympathetic inhibition (i.e. muscle relaxation) (Wu et al. 1999). Reflex-induced reductions in sympathetic drive can contribute to overall relaxation by decreasing muscle tone, heart rate and blood pressure (van den Berg and Cabri 1999; Wu et al. 1999). Small amplitude SS had no significant inhibitory effect on the E-reflex (Delwaide et al. 1981), whilst larger ROM stretches decreased both H- and E-reflexes similarly (Guissard et al. 2001). Nonetheless, this depression lasted only several seconds after stretching was ceased, and therefore should not directly contribute to ongoing losses of activation detected minutes after stretch cessation. Fowles et al. (2000) speculated that types III (mechanoreceptor, e.g. pressure, vibration) and IV (nociceptor, i.e. pain, temperature) afferents might also contribute to motoneurone inhibition. According to the results of Guissard and Duchateau (2006) in humans as well as in animal (cat) studies (Cleland et al. 1990; Cleland and Rymer 1990; 1993), joint and cutaneous receptors have not been found to provide strong inhibition of the motoneurons during small-amplitude stretches and play only a small role with larger amplitude stretches. Hence similar to the H-reflex, E-reflex

inhibition may occur during large amplitude stretches but desist soon thereafter and thus would not substantially contribute to subsequent performance impairments.

Golgi Tendon Inhibition

Stretching to, or near the point of, discomfort is typically perceived to place considerable stress and strain upon the musculotendinous unit. Since Golgi tendon organs (GTO; type Ib afferents) respond to musculotendinous tension (Houk et al. 1980), it could be assumed that the tension might induce GTO inhibition and thus promote muscle relaxation (i.e. lower muscle tone). The autogenic inhibition (GTO reflex) reflex is di-synaptic, inhibiting the same muscle group that provided the tension (Khan and Burne 2009). Walshe and Wilson (1997) suggested that increasing musculotendinous unit stiffness might increase GTO inhibition. However, Edin and Vallbo (1990) monitored microneurography, EMG and torque signals during ramp and hold stretches and reported that GTOs (Ib afferents) were insensitive to the stretch-induced tension, even though muscle spindle afferents (Ia and II) responded rapidly to stretch. However, as for the effects of stretch-induced force, GTO effects persist for only approximately 60-100 ms after cessation of stress, the duration of these inhibitory effects does not appear to be sufficient to influence subsequent muscle performance (Houk et al. 1980; Trajano et al. 2017).

Corticospinal excitability / inhibition

Stretch-sensitive muscle spindles are innervated by both type I and II sensory (afferent) neurones (Prochazka and Ellaway 2012). These fibres project to structures within the brain such as the somatosensory and the primary motor cortices (Phillips et al. 1971; Rathelot and Strick 2009). Additionally, afferent neurones innervating skin and joint receptors, and which are

activated during stretch, project to the somatosensory cortex and thalamus, which could also influence motor output from the primary motor cortex (Canedo 1997). Therefore, it has been speculated that changes in cortical circuits could be involved in the force loss observed after moderate-duration passive stretch (Trajano et al. 2017).

To date several studies have used transcranial magnetic stimulation (TMS) to investigate the possible effect of stretching on motor cortex excitability and the strength of its projection to motoneurons with conflicting results. When applied to the motor cortex at an intensity above motor threshold, TMS elicits a motor evoked potential (MEP) in the target muscle that is reflective of corticospinal excitability. Studies that have measured MEP amplitudes when the muscle is at rest (no voluntary contraction) after passive stretching have reported no significant changes, suggesting a lack of change in corticospinal excitability (Budini et al. 2017; 2019; Pulverenti et al. 2019). In particular, Pulverenti et al. (2019) reported a significant reduction in maximal force after stretching without reductions in MEP amplitude and, perhaps more importantly, no associations between individual variations in MEP responses and the magnitudes of reduction in maximal force. However, several studies have measured post-SS MEP amplitudes during voluntary contractions of various intensities and obtained different results. MEPs were unchanged when measured during contractions at maximum (Pulverenti et al. 2019) and 20% of maximum (Pulverenti et al. 2020) after 5 min (5 sets of 60 s) of intense passive stretching, but increased when measured during a 30% contraction after 100 s (5 sets of 20 s) passive stretching (Opplert et al. 2020). It is difficult to reconcile these findings because different stretching protocols and measurement methods were used, but the current evidence indicates that SS has minimal effect on corticospinal excitability, and certainly does not appear to reduce it. Thus,

there is currently no evidence to support the hypothesis that SS-induced force reductions might be explained by a reduction in corticospinal excitability.

The cortical silent period (**an interruption of voluntary muscle contraction**) observed when a single TMS pulse is applied to the motor cortex during a voluntary contraction, might also reveal cortical mechanisms underpinning stretch-induced force loss. This silent period duration is believed to be largely caused by the activation of gamma aminobutyric acid (GABA) inhibitory interneurons within the motor cortex by the TMS pulse and is usually indicative of intra-cortical inhibition. Three studies have measured the cortical silent period after SS, but no changes were detected during maximal contractions (Pulverenti et al. 2019) or those performed at 20% (Pulverenti et al. 2020) or 30% (Opplert et al. 2020) of maximum. These findings suggest that intra-cortical inhibition, measured as the cortical silent period, is not affected by passive stretching and is not currently a candidate explaining post-SS force losses.

Motoneurone Responses

In addition to the Na^+ -based current inflow that occurs as action potentials traverse the motoneurons, the dendritic regions of motoneurons have an important intrinsic mechanism for amplifying and prolonging the normal synaptic input they receive (e.g. from supraspinal centres and reflex pathways) by allowing a persistent inward current (PIC) flow through specialised Na^+ and Ca^{2+} channels **resulting in sustained depolarization** (Binder et al. 2020). These PICs are greatly enhanced in the presence of monoamines such as serotonin and noradrenaline, which are released in the spinal cord by nerves emanating from brainstem nuclei, and are capable of enhancing motor output by up to 5-fold (Lee and Heckman 2000). This amplification of normal drive to the muscle is in fact fundamental to the capacity for motoneurons to fire at the high

frequencies necessary to produce maximal and near-maximal levels of force. In the absence of these PICs, and neuromodulatory input from serotonergic and noradrenergic neurones, motoneurones would scarcely produce 40% of their maximal normal output (Heckman 1994). Therefore, PICs play an essential role in normal motor behaviour and particularly in our ability to produce rapid and high levels of muscular force.

One hypothesis that has been recently advanced (Trajano et al. 2017) is that SS might affect PIC-dependent amplification of central drive to the muscle, which would consequently reduce maximal force capacity. Indirect evidence to support this hypothesis originated from an experiment in which tonic vibration reflexes (TVRs) caused by tendon vibration were elicited before and after 5 min of passive stretching (Trajano et al. 2014b). TVRs elicit involuntary contractions via steady stimulation of the Ia reflex loop. The contractions evoked by bursts of neuromuscular electrical stimulation (NMES; 5×2-s bursts, 20 Hz frequency, ~20% MVC) superimposed over the tendon vibration display features that are consistent with the initiation of PICs, including ongoing (i.e. self-sustained) motor unit firing after both vibration and NMES are ceased, inhibition of responses when the antagonist muscle is at long muscle length (inducing reciprocal inhibition), and the continuous increase in muscular force in response to the repetitive activation (i.e. warm-up effect) (Binder et al. 2020). Interestingly, all of these parameters were reduced immediately markedly after 5 min of passive stretching, partially recovered by 5 min, and fully recovered by 10 min after stretching (Trajano et al. 2014b). This time course of both reduction and recovery closely matches that which is observed in other experiments with similar stretching protocols (Pulverenti et al. 2019; 2020; Trajano et al. 2014a). These data provide some early evidence that stretch-induced PIC reductions might at least partly underpin the loss of force, and their recovery might be also important for the recovery of force. However, additional

experiments with more robust, or direct, techniques are necessary to confirm this hypothesis. In the absence of other candidates for the post-SS loss of neural drive, assessments of the effects of SS on PICs are clearly warranted.

Summary of neural mechanisms

As summarised in Table 2, although many stretching studies attribute performance impairments to neural inhibition, current evidence is inconsistent. While tonic reflexes may increase muscle tone and contribute to resistance to movement, SS effects on EMG activity is conflicting, with both decreases and no change reported. Alterations in neuromuscular activation as measured by EMG activity may be obscured by peripherally induced modifications to the muscle action potential as well as the curvilinearity of the EMG-force relationship. Hence, EMG and reflex activity normalized to the M wave tend to present a more consistent decrement with stretching. Whereas inhibitory inputs from proprioceptive structures (i.e. H-reflex: Ia afferents and Golgi tendon organ: Ib afferents) can reduce facilitatory reflex activity, the duration of their effects tends to be too transient to meaningfully affect subsequent performance. However, stimulation of exteroceptive reflexes may aid in the suppression of excitatory sympathetic nervous activity contributing to a more relaxed state and less resistance to movement. The literature to date generally indicates that corticospinal excitability and intracortical inhibition (silent period) are not substantially affected by stretching, however reduced strength of PICs at the motoneurone dendrites could be adversely affected and contribute to force output deficits. Thus, despite the considerable evidence for reductions in neural drive underpinning the stretch-induced force loss, the exact mechanism/s causing the effect remain elusive.

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Morphological responses to static stretching

Muscle and tendon stiffness alterations

As the neuromuscular responses to stretch vary between studies and may not completely explain the immediate (Kay and Blazevich 2008; McBride et al. 2007; Power et al. 2004) or prolonged (Fowles et al. 2000; Trajano et al. 2014a) reductions in force after stretching in all cases, alternative mechanical and/or physiological mechanisms associated with post-SS force losses should be considered. One hypothesis is that changes in the stiffness of either contractile or passive elastic elements within the MTU may compromise force transmission and/or alter *in vivo* sarcomere dynamics during contraction, and thus impact the external force produced. For some joints, reductions in the slope of the passive moment-angle curve, which are considered to reflect changes in whole MTU stiffness, have been reported after acute SS (Kay and Blazevich 2009b; Kubo et al. 2001; Magnusson et al. 1996a), as reported in our previous review (Behm et al. 2016a). Since 2016, a further 14 studies have examined the acute effects of SS on force output and MTU stiffness. Six studies targeted the knee flexors and all reported significant reductions in MTU stiffness, however only studies imposing longer duration (3-5 min) stretches reported concomitant reductions in peak isometric force production (Hatano et al. 2019; Kataura et al. 2017; Matsuo et al. 2019), with no reduction in peak isometric (Palmer et al. 2019; Palmer and Thiele 2019) or concentric (Takeuchi and Nakamura 2020) force following shorter duration (20-120 s) stretches. Similar findings were reported in the plantar flexors, with seven studies reporting reductions in MTU stiffness but with reductions in force generally reported only after longer-duration (2.5-5.0 min) (Bouvier et al. 2017; Konrad et al. 2019; Longo et al. 2017;

Opplert et al. 2016) but not shorter-duration (2-3 min) stretches (Konrad et al. 2017a,b; Konrad and Tilp 2020a). Also, one study reported reductions in MTU stiffness lasting at least 10 min but reductions in force lasting only 5 min (Konrad et al. 2019), and another study employed shorter-duration stretching (60 s) and reported no change in stiffness or muscle force (Konrad and Tilp 2020b). Finally, to our knowledge no studies directly assessed the relationship between changes in stiffness and changes in muscle force production, so a direct stiffness-muscle force link has not been established. These findings are consistent with the research conducted before 2016, in which only longer durations of stretch were associated with both peak force and MTU stiffness reductions and that associations between changes in MTU stiffness and muscle force after SS were weak. Thus, the data conclusively show that the factors influencing passive whole MTU stiffness do not substantially influence maximal voluntary muscle force production in the muscles studied. This makes sense given that (i) active and passive stiffness are unrelated when measured in *ex vivo* experiments (Prado et al. 2005), (ii) active muscle stiffness (i.e. responses to stiffness properties measured in humans during dynamic muscle contraction, e.g. by measuring oscillation responses to brief perturbations) is not related to passive MTU stiffness measured in muscle groups such as the ankle plantar flexors (Hunter and Spriggs 2000) or knee flexors (Blackburn et al. 2004), and (iii) reductions in passive MTU stiffness have been observed without changes in active stiffness after an acute bout of static plantar flexors stretching (Hunter 2001). The effects of stretching on active muscle mechanical properties after SS, therefore, cannot be inferred from measurements of passive stiffness.

Nonetheless, alterations in whole MTU stiffness may be negligible even when decreases in muscle (and fascicle) stiffness are clearly detectable (Blazevich et al. 2012a; Konrad et al. 2019). Several studies have detected changes in plantar flexor muscle stiffness, measured as the

change in the passive muscle force-length relationship, without changes in tendon stiffness after 1-5 min of SS (Kay and Blazevich 2009a; Kay et al. 2015; Konrad et al. 2017a;b; Morse et al. 2008; Nakamura et al. 2011); however, it is not yet clear how stiffness is altered in other muscles, and no studies examined relationships between changes in muscle stiffness and changes in force production. Nonetheless, a reduction in tendon stiffness was observed when longer SS durations were used (10-20 min)(Kato et al. 2010; Kubo et al. 2001), suggesting that the tendon may be influenced by long stretch durations, although this may be influenced by the measurements being taken at the fascicle-aponeurosis junction which then allows changes in muscle stiffness to possibly contribute to the changes in ‘tendon’ stiffness. Tendon stiffness decrease may be expected to reduce muscle forces by reducing muscle length during contraction (Mayfield et al. 2016) or inducing a shortening-induced force depression, i.e. reduction in the force produced at a given muscle length that results from shortening immediately prior to the muscle reaching that muscle length (Raiteri and Hahn 2019). However, since short- and moderate-duration stretching do not significantly alter tendon stiffness, these phenomena are unlikely to be relevant to the stretch-induced force loss. Furthermore, post-SS force losses may be observed even when the muscle generates tension at the same length after stretching (Kay and Blazevich 2009b), yet force losses are not always observed even in cases where tendon stiffness is reduced (Kubo et al. 2001). Therefore, whilst changes in muscle-specific stiffness may temporally relate to changes in force production, a direct link has yet to be shown and changes in tendon stiffness do not appear to be associated with muscle force reductions. Thus, whilst further research is required to investigate possible links between changes in passive muscle mechanical properties and changes in active force production after stretching, there is little current evidence that changes in MTU stiffness directly influence the post-SS force decrement.

Intramuscular connective tissue responses

Passive and active muscle stiffness measurements reflect mechanical properties of very different structures. As the parallel elastic component (PEC; including the endo-, peri- and epimysia) is literally in parallel with the contractile component, it is commonly considered to be unloaded during muscle contraction (MacIntosh and MacNaughton 2005; Rode et al. 2009). Thus, its contribution to muscle stiffness during active muscle contraction should be negligible. However, the PEC plays a significant role in resisting muscle lengthening during passive stretch (Gillies and Lieber 2011; Prado et al. 2005). Muscles contain a large amount of collagenous perimysial tissue in particular (Purslow 1989), which is thought to contribute significantly to the muscle's stretch resistance (Borg and Caulfield 1980; Purslow 1989; Williams and Goldspink 1984). Given that muscle stretching may acutely reduce passive muscle stiffness, it is likely that stretching affects the mechanical properties of the PEC itself. Consistent with this, both reduced MTU stiffness and increased electromechanical delay (EMD) have been concomitantly observed after SS (Costa et al. 2010; Hirata et al. 2016; Taniguchi et al. 2015). EMD comprises both electrochemical and mechanical components (Rampichini et al. 2014), and although lengthening of both components is reported immediately following SS (Esposito et al. 2011; Longo et al. 2017), the electrochemical component appears to recover rapidly whereas the mechanical component, thought to strongly reflect PEC stiffness, often remains impaired for many minutes after stretch and is consistent with the temporal recovery of MTU stiffness (Esposito et al. 2011). Nonetheless, only indirect evidence for the effect of stretching on the PEC has been presented, so it is not yet explicitly clear whether stretching can trigger changes in the PEC that persist after the stretch is completed.

Any change in PEC stiffness might be functionally relevant. Although changes in PEC stiffness should not directly affect muscle force by reducing the passive component of the total force (i.e. total force = passive + active) under most conditions (MacIntosh and MacNaughton 2005; Rode et al. 2009), there are two potential mechanisms by which PEC stiffness might influence muscle force. First, evidence exists in both animal models (Huijing 1999; Huijing et al. 2007) and in human muscles (Bojsen-Moller et al. 2005) that forces produced within muscle fibres are transferred to the skeleton via the PEC. Reductions in PEC stiffness could therefore affect the efficiency of force transmission within the muscle, and to the external tendon and skeleton (Huijing 1999; Huijing and Baan 2003). These possibilities are yet to be explicitly explored. Second, axial (longitudinal) fibre shortening during contraction is also accompanied by radial fibre (transverse) expansion since muscles (and fibres) are relatively isovolumetric during contraction (Baskin and Paolini 1967). Therefore, some parts of the PEC must be stretched during muscle shortening (including during ‘fixed end’ isometric contractions) and thus its properties may influence shortening capacity (Roberts et al. 2019). Also, fibres in most muscles are not arranged in line with the muscle’s longitudinal axis but are oriented at an angle (the pennation angle), so stretch and shortening of muscle may cause some stretch/shortening in the PEC, and thus the fibres and muscle, both axially and radially. Both axial and radial forces will influence the amount of fibre rotation during contraction and hence the ratio of muscle-to-fibre length change (or velocity) (Eng et al. 2018; Roberts et al. 2019). This ratio, i.e. the “gear” in which the muscle operates, influences muscle force and velocity characteristics (increased rotation [i.e. gear] increases velocity at peak power). Current evidence suggests that the mechanical properties of intramuscular connective tissues contribute to the magnitude of fibre rotation (Eng and Roberts 2018; Holt et al. 2016), and thus the muscle’s gear, and therefore must

influence the force (and power)-velocity properties of the muscle (Roberts et al. 2019). Thus, based on this current view of the role of the PEC, changes in the PEC in response to muscle stretching may theoretically influence active muscle force production. However, it is not clear whether these would increase or decrease muscle force production, or whether it would vary depending on the contraction type (e.g. isometric/concentric vs. eccentric vs. stretch-shorten cycle), shortening velocity or external load. These concepts should thus be foci of future research.

Length-tension effects

The lack of change in tendon stiffness after short- and moderate-duration stretching implies that the muscle should function at the same length for a given level of muscle force after stretching; thus shifts in the force-length (length-tension) relation might not be expected to underpin force losses. Nonetheless, several studies have confirmed that the active length-tension relationship is shifted towards longer lengths (i.e. larger joint angles; rightward shift) after SS (Cramer et al. 2007; Takeuchi and Nakamura 2020; Weir et al. 2005). This is important as many studies test the impact of SS during contractions (often isometric) at relatively short muscle lengths; in fact, all studies on the human plantar flexors are likely to have tested on the ascending limb of the force-length relation, and thus at a ‘short’ muscle length (Herzog et al. 1991; Maganaris 2003). Therefore, the results of many studies would be vulnerable to a rightward shift that would manifest as a reduced maximal force. In fact, studies in which tests were conducted at several muscle lengths reported post-stretch strength losses at short muscle lengths but moderate improvements at the longest muscle lengths tested (Balle et al. 2015; Herda et al. 2008; McHugh and Nesse 2008; McHugh et al. 2013; Nelson et al. 2001a). Collectively, these data support a

rightward shift in the length-tension relation and suggest that at least some of the force loss measured in previous studies might be explained by this mechanism. Future studies might circumvent this issue by testing muscle function at several muscle lengths or reporting muscle length-specific forces (or angle-specific joint torques) during dynamic muscle contractions.

The question also arises as to the mechanism underpinning the shift in the force-length relation, given that reductions in tendon stiffness, which would tend to reduce the muscle length for a given force level, are uncommon after short- and moderate-duration stretching. One possibility is that PICs (described above) are more affected at some joint angles, and thus muscle lengths, than others, altering muscle activation in a muscle length-dependent manner. PICs are known to be joint angle dependent, partly because altering the length of the antagonist muscle influences reciprocal inhibition onto the agonist muscle (Gorassini et al. 2002) and partly because the influence of PIC function on agonist force production is muscle length dependent (Kim 2017). Therefore, PICs in agonist muscles should be stronger at longer muscle lengths, and thus potentially less susceptible to post-stretch attenuation. However, this hypothesis is yet to be tested. An additional neural hypothesis is that muscle stretching may reduce inhibitory afferent responses that would otherwise act to reduce neural drive to muscles held at longer lengths. The effect of muscle length on inhibitory gain can be readily observed in H-reflex measurements, whereby a reduction in H-amplitude (i.e. greater inhibition) is observed as agonist muscle length is increased in muscle groups such as the plantar flexors (Blazevich et al. 2012a; Guissard et al. 1988; Mark et al. 1968) inhibitory feedback through reciprocal, cutaneous, recurrent, and presynaptic inhibitory pathways strongly reduces H-reflex amplitudes (Blazevich et al. 2012b; Crone et al. 1990; Day et al. 1984) so a greater reduction in H-amplitude at longer lengths is suggestive of greater inhibition. However, while it is known that acute muscle stretching can

reduce H-reflex amplitudes, possibly indicating an increase in inhibitory feedback onto the agonist motoneurone pool, the muscle length-dependence of this effect has not been examined to our knowledge. Thus, this hypothesis remains to be properly tested.

Alternatively, length-dependent changes in force production may be triggered by changes in calcium sensitivity of the acto-myosin complex, i.e. a 'fatigue-like' effect. Calcium sensitivity is decreased by factors that reduce the total cross-bridge force for a given amplitude of calcium release (this amplitude is a function of the central, efferent drive to the muscle), including accumulation of metabolic by-products (H^+ ions, inorganic phosphate, adenosine diphosphate, lactate ions and others) or reactive oxygen and nitrogen species (ROS/RNS) as well as increases in muscle temperature (Stephenson and Williams 1985), reductions in intracellular water (Edman and Andersson 1968; Sugi et al. 2013), or dephosphorylation of the myosin regulatory light chain (Vandervoort et al. 1983). Such effects would be consistent with the significant reductions in muscle twitch force usually (Behm et al. 2001; Costa et al. 2010; Opplert et al. 2016), although not always observed after SS (Opplert et al. 2020), given that muscle twitch forces are considered to be highly sensitive to changes in calcium sensitivity (Moore and Stull 1984; Vandenoorn et al. 2013). They may also help to explain reductions in electrically-evoked tetanic forces after prolonged stretch (e.g. 5 min)(Trajano et al. 2014a). It is interesting to consider that the ongoing metabolism of muscle during the ischaemic period of the muscle stretch may trigger the accumulation of metabolic by-products or reactive oxygen or nitrogen species and thus contribute to a loss of calcium sensitivity and a stretch-induced force loss. If stretching were to negatively affect contractile dynamics through a reduction in calcium sensitivity then these effects could be (at least partly) overcome by moving the agonist muscle to a longer length, which increases calcium sensitivity (Balnave and Allen 1996; Stephenson and

Wendt 1984), and this might partly explain the length-dependence of post-SS force loss. The effects of stretching are not well understood in this context, although it is known that passive muscle stretching can increase ROS/RNS production in skeletal muscle, either as a direct effect of the stretch (Chambers et al. 2009; Palomero et al. 2012; Tidball et al. 1998) or through reduction in muscle circumference during stretch. This increases intra-muscular pressure to expel blood from, and prevents arterial (oxygenated) flow into, the muscle (Otsuki et al. 2011). An increase in ROS/RNS production can reduce calcium release from the sarcoplasmic reticulum, impairing excitation-contraction coupling and consequently reducing muscle force (Bruton et al. 2008; Lamb and Westerblad 2011). In humans, Trajano et al. (2014b) found that continuous plantar flexors stretch (1 set of 5 min) produced greater ischemia, estimated using near infra-red spectroscopy, than intermittent stretching (5 sets of 1 min; 15-s interval), however the multiple cycles of ischemia and reperfusion during intermittent stretching might be expected to cause greater ROS production (Blaisdell 2002; Gute et al. 1998; Powers and Jackson 2008). The greater magnitude and longer duration (at least 30 min) of force loss after intermittent SS observed by the researchers is consistent with this hypothesis, however no evidence of impairment of the excitation-contraction coupling process was detected during electrically-stimulated contractions, so other mechanisms must have impacted muscle function. Therefore, although it is possible that an increased production of ROS/RNS or other metabolic products is involved in the force loss observed after (particularly intermittent) stretching, there is currently a lack of direct evidence in humans.

Architecture and gearing effects

Alterations in muscle blood flow, pressures or connective tissue (intramuscular or tendinous) properties might theoretically influence the muscle's architectural organisation, and particularly fibre (pennation) angle. Increases in pre-contraction fascicle angle may reduce the proportion of fibre force directed along the tendon and thus total muscle force. Also, any change in the rotation of fibres (and thus the gear ratio adopted) during contraction may influence the force (and power)-velocity profile of a muscle (Eng et al. 2018). However, studies employing the requisite imaging technologies have reported no change in resting pennation angle or fascicle length following SS (Ce et al. 2015; Kay and Blazevich 2009a;b; Opplert et al. 2016), despite reductions in force and stiffness. Furthermore, where a reduction in whole-muscle operating length has been reported (Kubo et al. 2001) after longer SS durations (10 min), no reduction in maximal voluntary force was observed. Finally, no studies have examined fascicle rotation (i.e. gearing) during contractions before and after stretching, so it is not yet known whether stretching alters dynamic fibre or fascicle behaviour. Therefore, there is no current evidence that SS-induced alterations in muscle architecture directly influence muscle force production. However, dynamic fascicle behaviour has not been examined, so it is unclear whether fascicle rotation during contraction is altered by acute muscle stretching.

Titin

Passive muscle stiffness has been suggested to be largely attributable to adaptations in collagenous components embedded within the extracellular matrix (ECM) of endo-, peri-, and epimysial connective tissues (Ward et al. 2020), with titin filaments acting as a molecular spring responsible for almost all passive force within the myofibril (Herzog et al. 2012). However, more recent work in animal models has suggested that titin may still play an important role in

determining stiffness at the whole muscle level (Brynnel et al. 2018), thus potential SS-dependent changes in titin mechanics may theoretically affect its contribution to whole muscle stiffness and force production. The elimination of titin from the myofibrils has been demonstrated to abolish all passive and active force in myofibrils, indicating the importance of titin not only for passive force production but also for active force transmission within the sarcomere (Leonard and Herzog 2010). More recently, direct force production by titin has been shown through folding of its Ig domains (Rivas-Pardo et al. 2016). Titin can also contribute to active force during eccentric contractions through calcium binding to titin (to increase titin's inherent stiffness) and by titin binding to the actin filament, thereby reducing titin's free spring length to increase its stiffness and force contribution (Rassier et al. 2015)). Importantly, titin's stiffness, and therefore its contribution to force at a given sarcomere length, is thought to be influenced by muscle length during contraction initiation or by passive stretch (Herzog 2014; Leonard and Herzog 2010). When contractions are initiated at shorter muscle lengths, titin is thought to bind on the actin filament further from the z-line, which would decrease the length of the free part of the titin filament (Herzog et al. 2012) and increase titin's stiffness. However, during passive stretch titin fails to bind to actin increasing its spring length and decreasing its stiffness. Furthermore, as no contraction is initiated during passive stretch, calcium is disassociated from titin further reducing its inherent stiffness. While changes in titin stiffness could theoretically contribute to the temporary post-stretch reduction in muscle stiffness, increased slack length, rightward shift in the length-tension curve, and maximal force reduction, the effects could possibly be very short lasting - altered titin stiffness may persist for only a few seconds after stretch or contraction is terminated (Lee et al. 2007). Thus, there is no current evidence that longer-term changes in titin properties (i.e. minutes after stretch) might produce an

ongoing effect on muscle force. Further research is required to fully elucidate whether a titin-based mechanism contributes to post-SS force losses.

Summary of morphological mechanisms

As summarised in Table 3, reductions in MTU stiffness and maximal force output are commonly reported following longer duration stretches, with force reductions primarily occurring at shorter muscle lengths with no change or slight increases at the longest muscle lengths. However, changes in MTU stiffness are not temporally associated with change in muscle force production and are therefore unlikely to directly influence force. It is also unlikely that changes in tendon stiffness (which might then reduce active muscle length) are sufficient to affect muscle force. Nonetheless, alterations in passive muscle force, possibly indicating a reduction in stiffness of the muscle's parallel elastic components, is commonly observed. Theoretically, changes in the PEC may influence muscle force by (i) impairing force transmission at several levels of the muscle's hierarchy, (ii) reducing the radial (transverse) component of passive fibre forces that is axial (longitudinal) to the line of action of the muscle, and (iii) reducing fibre/fascicle rotation during contraction (including in fixed-end isometric contractions, during which tendon stretch allows for muscle shortening) and thus altering both the muscle's gear ratio and line of pull of fibres. However, these possibilities have yet to be explicitly studied in relation to the post-SS force loss. Notably, SS tends to promote a rightward shift in the force length curve; factors that might underpin the shift, including reduction of PIC strength (which is joint angle-dependent), length-dependent inhibition of the motoneurone pool, and reduced Ca^{2+} sensitivity (e.g. through metabolite accumulation) could all impact muscle force production but have also received little attention to date. Finally, changes in the properties

of titin may speculatively influence muscle force, however this possibility has yet to be studied. Importantly, the current literature reveals that some mechanisms often theorised to underpin post-SS reductions in force are often not detected, poorly related to changes in force, or somewhat speculative and remain to be examined.

PLACE TABLE 3 APPROXIMATELY HERE

Psychological Influences

Mental energy deficit

Prolonged SS at or near the point of discomfort can be uncomfortable and it may require concentration (cognitive demand) for maintenance of joint position and stretch intensity. If this triggers some level of mental fatigue then this might have some impact on performance, especially in repetitive or prolonged, continuous activities (Marcora et al. 2009; Pageaux et al. 2013; 2014). Mentally fatiguing tasks cause individuals to perceive a subsequent task to be more taxing or demanding, and thus they may cease the activity earlier or provide less effort (Marcora et al. 2009; Pageaux et al. 2013; 2014). This mental energy deficit is a global body phenomenon as performance impairments have been demonstrated in non-exercised homologous and heterologous muscles following fatiguing protocols of a contralateral muscle that necessitated prolonged concentration to maintain the fatigue-inducing forces (Halperin et al. 2015). Furthermore, the stretching of one muscle group can enhance the ROM of other homologous (Chaouachi et al. 2017) and heterologous (Behm et al. 2016b) muscle groups. This global effect of SS may speculatively be attributed to the concept of increased stretch tolerance (Magnusson et al. 1996b; 1997). The outcomes of studies on the effects of unilateral SS on contralateral limb performance are

conflicting, with impairments in knee extensor MVC force (4.2%)(Caldwell et al. 2019), rate of force development (10.8%)(Jelmini et al. 2018), voluntary activation (interpolated twitch technique: 7%) and mechanomyogram (9%)(Ce et al. 2020) contrasting with no contralateral decrements in knee extension MVC (Behm et al. 2019) or isokinetic torque or power (Chaouachi et al. 2017). Hence if a psycho-physiological phenomenon such as stretch tolerance can induce global increases in ROM, with further evidence, albeit not unanimous for SS-induced contralateral strength and activation deficits, it is likely that a prior mentally fatiguing task such as prolonged SS at or near the point of discomfort could contribute to global performance impairments. As the literature is not extensive concerning non-local stretching effects, more research is needed.

Placebo and Nocebo effects

The review by Behm et al. (2016a) highlighted a number of limitations within current research, one being the possibility of placebo or nocebo effects that could influence the performance of participants who were familiar with the literature relating to the effects of SS. In that context, Janes et al. (2016) demonstrated that participants who were deceived to believe that prolonged SS was beneficial showed increases in knee extension MVC force (5.1%-8.8%). Similarly, Bertolaccini et al. (2019) used positive and negative biased groups as well as a control group. There were no differences in total number of repetitions performed during a strength endurance test (4 sets to failure at 70% of 1 repetition maximum (1RM)), but the positively biased group performed a greater number of repetitions in the final set. Blazeovich and colleagues (2018) incorporated 30 s of SS into a full warm-up and found improved ROM but no deficits in jumping, sprinting or agility tests. Participants were queried about their performance expectations when stretching was included. Prior to the experiment, 18/20 participants indicated

that dynamic stretching was most likely to improve performance and 15/20 participants nominated no stretching to be least likely. However, there was no relation between their ratings and test performances. Nevertheless, the authors reported evidence that including SS or dynamic stretching into the warm-up instilled more confidence in their subsequent athletic performance. Hence, the body of literature on placebo or nocebo effects of stretch perceptions is sparse. But since psychological effects play a vital role for optimal performance, and appropriate durations of SS and dynamic stretching within a full warm-up generally do not impair performance, short to moderate durations of SS (e.g. <60 s) within a comprehensive warm-up can be generally recommended.

PLACE TABLE 4 APPROXIMATELY HERE

Conclusions

Various forms of stretching have been used for millennia in preparation for subsequent activity (acute), training to enhance ROM (chronic), rehabilitation, psycho-physiological relaxation (i.e. yoga), and other scenarios. Evidence has accumulated over approximately the last 25 years for SS-induced impairments with prolonged acute stretching when performed without a complement of dynamic warm-up activities or with little time between the end of muscle stretching and commencement of physical activity. Mechanisms underlying acute performance impairments under these conditions have been attributed to neural, morphological and psychological factors. While SS-induced EMG decrements are inconsistently reported in the stretch literature, EMG normalised to the M-wave provides more consistent evidence for neural deficits. Reflex inhibition (i.e. H-reflex, E-reflex, GTO) from SS tends to dissipate quickly after

SS. The literature illustrates minimal effects of SS on corticospinal excitability or the cortical silent period, but motoneurone excitability can be adversely affected by stretch-induced alterations in the strength of persistent inward currents (PICs), which may persist up to 10 min after stretching; both the temporal profile of PIC changes and their importance for achieving maximal muscle activation implicate this mechanism as a probable primary driver of SS-induced force losses. Regarding possible morphological effects, shifts in the active force-length relationship appear to be associated with force changes. They may impact directly, as a result of increasing (at longer lengths) or decreasing (at shorter lengths) the maximal force production capacity. However, the shift may also implicate other mechanisms, such as reductions in PIC strength (which are joint angle-dependent), inhibitory feedback onto the motoneurone pool, or Ca^{2+} sensitivity (associated with metabolite accumulation; i.e. ‘fatigue-like effects’) as possible mechanisms influencing muscle force production. Nonetheless, direct examination of these possibilities is needed before conclusions can be made as to their direct importance. Alterations in PEC stiffness may also be evoked by SS, and while there is not expected to be a direct impact on muscle force (since the PEC is largely unloaded during contraction), indirect effects on force transmission efficiency, decreased off-axis passive force contributions or alterations in fibre rotation during contraction (i.e. ‘gearing’; including during fixed-end isometric contractions) may influence force production. Again, however, little direct evidence of these mechanisms is available. Also, while effects of SS on titin function/properties are speculatively possible, no current evidence exists to determine possible effects. Finally, psychological factors such as the mental energy deficit arising from prior prolonged focus or concentration with stretching or placebo effects could also contribute to adverse performance outcomes following SS (Figure 1).

PLACE FIGURE 1 APPROXIMATELY HERE

Recommendations

Whilst appropriate durations of SS (<60 s per muscle group) within a full dynamic warm-up can improve ROM with trivial (positive or negative) performance effects, there may be other benefits. SS may be used, if an individual wishes, before physical activities as long as SS durations are moderate (e.g. < 1 min per muscle group), especially when performed as part of a complete, pre-exercise routine. It may also assist force production at long muscle lengths when performed immediately prior to a task. With regards to psychological benefits, short duration pre-exercise stretching may also provide an opportunity for team bonding (stretch as a group). SS can also work as a form of self-diagnosis; a way to check different regions of the body for unusual soreness or tightness either before or after training and competitions. Additionally, reflex-induced reductions in sympathetic drive can contribute to overall health and performance by decreasing muscle tone, heart rate and blood pressure (van den Berg and Cabri 1999; Wu et al. 1999), which may speculatively improve performance in accuracy sports such as shooting, archery and biathlon. If longer-duration stretches are necessary within a warm-up to achieve substantial ROM improvements in sports or activities that require it then some performance decrement may be expected if minimal time elapses between stretching and task performance (at least in tasks performed at short and moderate muscle lengths). In these cases mitigation strategies would be useful, however there is still a lack of understanding of the mechanisms underlying SS-induced force reductions, so it is not yet possible to describe an optimal suite of strategies for use. Further research is required, in line with the recommendations of the present review, in order to better understand the mechanisms impacting muscle force reduction after SS.

Acknowledgement

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Table Legends

Table 1: Sample of acute static stretching studies since 2016 that have investigated strength and power performance variables.

Acronyms: BB: basketball, CMJ: countermovement jump, Hams: hamstrings, HF: hip flexion, KE: knee extension, KF: knee flexion, MVC: maximum voluntary contraction, NR: not reported, NS: non-significant, POD: point of discomfort, RA: recreationally active, RT: resistance trained, RTime: reaction time, PT: peak torque, Quads: quadriceps, Sed: sedentary. Shaded boxes highlight studies that recruited youth.

Table 2. Summary of evidence for/against the effects of neurological factors on the SS-induced force loss.

Table 3. Summary of evidence for/against the effects of morphological factors on the SS-induced force loss.

Table 4: Summary of evidence for/against the effects of psychological factors on the SS-induced force loss.

Figure Legend

Figure 1: A) The effects of static stretching (SS) with and without a warm-up on performance. **B)** Mechanisms potentially underpinning the SS-induced performance impairment.

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Table 1: Sample of acute static stretching studies since 2016 that have investigated strength and power performance variables.

Acronyms: BB: basketball, CMJ: countermovement jump, Hams: hamstrings, HF: hip flexion, KE: knee extension, KF: knee flexion, MVC: maximum voluntary contraction, NR: not reported, NS: non-significant, POD: point of discomfort, RA: recreationally active, RT: resistance trained, RTime: reaction time, PT: peak torque, Quads: quadriceps, Sed: sedentary. Shaded boxes highlight studies that recruited youth.

Authors	n	Age (yrs)	Trained State	Prior aerobic warm-up	Stretching volume	Stretch Intensity	Stretched Muscles	Post-stretch Dynamic activity	Post-Test Time	ROM	Performance
Avloniti et al. 2016a	34M	20.5±1.4	Trained	8 min jog	10, 15, 20, 30, 40, 60s	POD	Hip adductors extensors, knee extensors, flexors, PF	NR	4 min	NR	15+20s stretch 10m sprint: 2.8% 20m sprint: 3.2% 10, 30, 40, 60s stretch Sprints: NS All durations: Agility: NS
Avloniti et al. 2016b	40M	21.2±1.6 20.8±0.8	Trained	8 min jog	1x20, 30, 40, 60s 2x10s, 3x10s, 4x10s, 6x10s	POD	Hip adductors extensors, knee extensors, flexors, PF	NR	3 min	NR	20s stretch both groups: 10 m sprint: 2.2-2.8% All other conditions NS
Balci et al. 2019	8M 8F	19.9±1.2	RA	No	30s 45s	NR	Lower limbs	NR	Imm	NR	CMJ 30s: +6.2%, d=0.19 45s: +10.5%, d=0.31
Barbosa et al. 2020	11M	21.5±2.4	Trained	3/5 conditions 10 min jog	30s	POD	Quads Hams PF	1/5 conditions 10 min jog	Imm	NR	CMJ WU: 1.5% d=1.0 SS: 1.5% d=1.0

											WU-SS: 1.5% d=1.0 SS-WU: 4.7% d=2.0
Begovic et al. 2018	8M 7F	27.1±3.7 26.9±3.9	Healthy	NR	10x30s	90-130 ⁰	Quads	NR	Imm	NR	Time delay EMG-Force 35.5%, d=0.21 -18.4%, d=0.14 7.5%, d=0.06
Behm et al. 2019	14M	19-30	RA	5 min cycle	8x30s (4 min)	POD	Quads Hams	NR	1 min	HF: 5.1% d= 0.74	KE MVC: -7.7% d= 0.26
Bertolaccini et al. 2019	18M	19-40	RT	5 min treadmill	3 exercises x 3 x 30s (2min 30s)	POD	Quads	NR	5 min	NR	Total volume: NS Positive bias: increased # reps in last set
Blazevich et al. 2018	20M	21.1±3.1	Athletes	Low intensity3 min jog, series of 5s high knees and butt kicks	1x5s, 3x10s (30s)	POD	Quads Hams Plantar flexors, Shoulders HF, Adductors Gluteals	Higher intensity2 min jog series of 5s high knees and butt kicks	7 min	NR	SJ, CMJ, DJ, 20m sprint, agility test: NS
Bouvier et al. 2017	11M	21.0±2.0	Trained	10 submax contractions	5x 30s (150s)	POD	Quads PF	NR	Imm	NR	MVC: -7.1 to -7.7%
Brusco et al. 2018	20M	18-28	Untrained	5 min cycle	30s	Mild discomfort	Quads Hams Glutes PF	NR	Imm	NR	CMJ: -3.2% D=0.16
Caldwell et al. 2019	22M 18F	23.6±7.5 21.7±2.5	RT	5 min cycle	4x30s (2 min)	POD	Hams	NR	1 min	NR	KE MVC: - -8.1% d= 0.3 DJ: +9.2% d=0.18

Ce et al. 2020	21M	22±3	RA	NR	5x45s (225s)	80-90% POD	Quads	NR	Imm, 5, 10 min	6-11%	KE MVC: -7 to -19%
Chatzopoulos et al. 2019	25M	11.8±1.6	Athletes TaeKwon Do	5 min jog	30s	Mild discomfort	Quads, Hams, Iliopsoas	NR	Imm	Hams: 5.6% d= 0.44	RTime: -1.7% d= 0.09 Force Sense -14.04% d= 0.22
Celebi et al. 2016	30M	21.5±3.7 18.7±0.5	Sed: 15 Soccer: 15	5 min cycle	20 min	POD	Quads	NR	Imm	Sed: 8.8% d= 0.37 Soccer: 5.1% d= 0.21	KE Isokinetic PT: NS
De Souza et al. 2019	11M 10F	22.5±2.1 25.6±5.1	Untrained	NR	3x30s (90s)	POD	Hams	NR	Imm, 1,3,5 min	NR	Hams PT Imm: -33.6% d=1.6 1 min: -12.5% d= 0.52 3 min: NS 5 min: NS
Fjerstad et al. 2018	20M 20F	22.5±1.8 23.6±4.2	Limited flexibility	Yes	NR	70% POD	Hip Adductors	NR	Imm	Passive:1.8% d= 0.21 Active: 2.2% d= 0.28	MVC Passive:3.4% d= 0.11 Active: 1.2% d= 0.04
Freitas et al. 2016	17M	22.1±2.7	Limited flexibility	NR	900s (LILD) 243.5 ±69.5s (HISD)	50% max passive torque 100% passive torque	Hams	NR	1, 30, 60 min	HISD ROM sig greater than LILD	NR
Hatano et al. 2019	11M 13F	20.5±1.1	Healthy	NR	300s	Maximum extension	Quads	NR	10,20, 30 min	NR	MVC: -1.8 to 2.5%
Ikeda et al. 2019	10M	22±2	RA	NR	15x1min (15 min)	Subtle pain sensation	Plantar flexors	NR	Imm, 15, 30, 60 min	Imm: 7.2% 15: 0.4% 30: 0.2% 60: -1.0%	PF torque -4.3% d= 0.26
Janes et al. 2016	28M	21.8±1.3 21.4±2.0	Biased:14 Deception:14	5 min cycle	3x30s (90s)	POD	Hams	NR	1 min 5 min	NR	KE MVC Biased: - -3.6 –

												-10.4%, d=0.06 Deception: NS
Kataura et al. 2017	9M 9F	20.6±1.2	Untrained	NR	180s	80%, 100%, 120% of pain onset	Hams	NR	Imm	80%: -0.2% 100%: 6.4% 120%: 7.8%	MVC 80%: NS 100%: -3.3 Nm Sig 120%: -2.9 Nm Sig	
Konrad et al. 2017a	9M 8F	24.9±4.2 23.3±2.5	Healthy	NR	4x30s (2 min)	95% max ROM	PF	NR	<5 min	15.4% d=0.72 21.9% d=1.24	MVC: -1.4, d=0.03 -3.3%, d=0.08	
Konrad et al. 2017b	21 M 4 F	23.3±2.5 23.4±3.7	Trained	NR	4x30s (2 min)	Max ROM	PF	NR	2 min	4.5% d=0.24	MVC: 0.3% d=0.08	
Konrad et al. 2019	7M 7F	27.5±8.3 24.9±3.1	Healthy	NR	5x60s (5 min)	Max ROM	PF	NR	Imm, 5, 10 min	16.5%, d=0.67 11.4% d=0.45 9.6%, d=0.47	MVC: -21.5%, d=0.54 -7.1%, d=0.21 -15.3%, d=0.36	
Konrad and Tilp 2020b	11M 3F	24.8±3.8 24.6±2.3	Healthy	NR	3x60s (3 min)	PF	PF	NR	Imm, 5 min	13.8%, d=0.43 11.3%, d=0.31	MVC: -4.3%, d=0.13 1.2%, d=0.03	
Konrad and Tilp 2020a	11M 14F	25.9±6.9 24.1±2.7	Trained	NR	2x30s (1 min)	Max ROM	PF	NR	Imm, 20, 40 min	Imm: 12.2% d=0.72 20 min: 10.8% d=0.67 40 min: 12.8% d=0.89	MVC Imm: -6.3% D=0.18 20 min: -6.2% d=0.17 40 in -7.5% d=0.22	

Lima et al. 2016	27F	23.8±1.8	15RT 12 ballet	2x10 m of knee hugs, lunges, walking toe touches,	3x30s (90s)	Maximum ROM	Quads Hams	NR	3 min	RT: 9.04% d= 0.43 Ballet: 2.7% d= 0.42	Quads and Hams PT: NS
Longo et al. 2017	18M	24.3±3.0	RA	NR	6x45s (4.5 min)	POD	PF	NR	Imm	16.6%, d=0.85	MVC -14.1%, d=1.1
Marchetti et al. 2019	15M	27.5±6.1	RT	5 min cycle	50%: 6x 40s (240s) 85%: 3x40s (120s)	50% 85% POD	Hams	NR	NR	50% POD: 4.6% d=0.55 85% POD 11.4% d=1.3	KF force -23.6%, d= 1.37
Martinez-Jimenez et al. 2019	42F 6M	32.1±7.6	Healthy	NR	Continuous: 1x2 min Intermittent 5x1 min	70% POD	Plantar flexors	NR	Imm	NR	Intermittent ML displacement 10.8% d= 0.36 Continuous 4.3% d= 0.09
Matsuo et al. 2019	16M	22.2±1.2	Healthy	NR	10x30s (5 min)	Tolerable without pain	Hams	NR	Imm	16.7% d= 2.2	KF MVC -14.3% d=0.61
Opplert et al. 2016	10M	24.0±1.5	RA	NR	1,2,3,4,10 x 30s	POD	PF	NR	Imm, 5 min		MVC: -5.4% RFD: -2.8%
Palmer et al. 2019	13F	21±2	RA	NR	1x30s 2x30s (60s) 4x30s (120s)	POD	Hams	NR	Imm	30s: 9.04% d=0.56 60s: 7.8% d=0.39 120s: 9.9% d=0.44	30s: -4.2% d=0.17 60s: +1.4% d=0.05 120s: +1.0% d=0.04
Palmer and Thiele 2019	11F	24±4	RA	NR	4x15s (1 min)	POD	Hams	NR	Imm		MVC: 0.6%, d=0.02 RTD: 4.8%, d=0.17

Reid et al. 2018	16M	27.6±2.2	RT	5 min cycle	30, 60, 120s	POD	Quads Hams	Dynamic stretch and activities	After warm-up, SS, DS/DA, 10 min	HF: 2.6%-4.7% d= 0.2-0.5 KF: 6.4-9.2% d= 0.3-0.4	Quad MVC -1.7 to -4.4% d= 0.1 – 0.2 VJ -1.8% - 1.1% d= 0.1
Saka et al. 2020	8M	21.6±2.3	Untrained	NR	15s	POD	Quads Hams PF Hip Adductor Hip Extensors	NR	Imm	NR	VJ: 12.1% d=1.6
Sato et al. 2020	20M	21±0.2	Non-athletic	5 min cycle	20-s	NR	Plantar flexors	NR	Imm, 5, 10 min	24.1% d=0.86	Conc/Ecc torque NS
Silva et al. 2018	13F	14.5±2.4	Athletes Gymnasts	5 min jog 5 min walk 8 CMJ	4 Hams 3 Adductor 2 Quads stretches of 90s each	Maximum ROM	Hams, Quads, Adductors	7 dynamic stretches	Imm	Change NR	CMJ: -8.1% d= 0.73
Stevanovic et al. 2018	12M	18±0.4	Athletes (BB)	6 min jog	6-min	NR	Lower limbs	8 min BB	Imm and after 8 min BB	NR	VJ Imm:-3.01% d=1.13 Post-BB: NS d= 0.26
Takeuchi & Tsukuda 2019	14M	20.2±0.7	Athletes (BB)	NR	5-min	NR	Quads Hams Plantar Flexors	NR	NR	Quads:1.8% d=0.26 Hams: 8.9% d= 0.52 PF: 5.7% d= 0.38	Quads VJ: NS Hams VJ: NS PF VJ: -3.3% d=0.34
Trajano et al. 2019	18M	26.8±4.5	RA	5 min cycle	Continuous 1x5 min Intermittent 5x1 min	Maximum tolerable	Plantar Flexors	NR	Imm, 15, 30 min	NR	RTD: Cont:-30.2% Inter: -15.1%
Vieira et al. 2019	14M	22.05±2.1	Trained	5 min cycle + Stretch	20, 40, 60s	POD	Hip and Knee Flexion	NR	Imm	NR	KE PT (60 ⁰ /s) 20s: -5.9% d= 0.49

											40s: -1.9% d= 0.14 60s: -4.1% d= 0.36
Descriptive Statistics (i.e. sums, means, frequencies) 42 studies	605M 218F 26.4% female	26.6±3.31 yrs Only 2 youth studies	18 studies: trained 8 studies: RA 13 studies: untrained or healthy 2 studies: limited flexibility 1 study: deception	21/42 included a warm-up (50%)	26/42 used >60s SS exclusively (61.9%)	25/42 used maximum intensity / ROM / POD exclusively (59.5%)	1/42 stretched upper body: shoulders (2.3%) All studies stretched lower body	5/42 used post-stretch dynamic stretches or activities (11.9%)	16/42 tested at 5 min or more post-stretch (38.1%)	8.04% mean increase in ROM 36 increases and 2 decreases in ROM (-0.2% and -1.0%) Mean effect size: d=0.60 (moderate magnitude)	1.5% mean decrease in performance measures. 40 negative and 23 positive measures. Mean effect size d=0.40 (small magnitude)

Table 2. Summary of evidence for/against the effects of neurological factors on the SS-induced force loss.

Neurological Factor	Likelihood	Notes
Voluntary activation		
-EMG	Unclear	EMG research is conflicting, with both decreases and no changes reported, which is most likely due to contrasting central and peripheral (i.e. M wave) influences as well as the curvilinear EMG-force relationship. As EMG is more variable, large but not small force reductions tend to show decreases in EMG.
-EMG / M wave	Likely	With EMG normalised to the M wave there is a consistent relationship with SS-induced force loss.
H-reflex: Ia afferents	Unlikely	Reduction in Ia and Ib reflex activity can occur during SS but desists rapidly following the stretch (transient post-stretch duration).
GTO: Ib afferents	Unlikely	
E-reflex	Possible	E-reflexes can suppress excitatory sympathetic nervous activity.
Corticospinal excitability (CSE)	Unlikely	Neither CSE or SICI are substantially affected by SS.
Short intracortical inhibition (SICI)		
Persistent inward currents (PICs)	Possible	SS might adversely affect PIC-dependent amplification of central drive to the muscle, consequently reducing maximal force capacity.

Δ: change in.

Table 3. Summary of evidence for/against the effects of morphological factors on the SS-induced force loss.

Morphological Factor	Likelihood	Notes
Whole MTU stiffness	Unlikely	No temporal association between Δ MTU stiffness and Δ force. Passive MTU stiffness not associated with active stiffness.
Passive muscle stiffness	Unclear	No data reporting Δ passive stiffness vs. Δ force. But Δ in mechanisms underpinning the Δ passive stiffness not expected to <i>directly</i> affect force.
Tendon stiffness	Unlikely	No temporal association between Δ tendon stiffness and Δ force.
PEC properties	Possible	Indirect evidence for decrease in PEC stiffness, and only theoretical evidence of effect on force.
Active force-length relation - Reduced PICs - Reduce (neural) inhibition - Reduced Ca ²⁺ sensitivity	Likely - Possible - Possible - Possible	Increase or decrease in force at specific muscle lengths (joint angles) commonly reported. Several mechanisms may influence shift, and also affect Δ force.
Altered architecture/gearing	Unclear	Little association between Δ fascicle angle and Δ force. Changes in fascicle rotation <i>during</i> contraction not yet studied.
Titin properties/function	Unknown	No research to date.

MTU: muscle-tendon unit; PEC: parallel elastic component; Δ : change in.



Table 4. Summary of evidence for/against the effects of psychological factors on the SS-induced force loss.

Psychological Factor	Likelihood	Notes
Mental energy deficit	Possible	Since stretch tolerance can induce global ROM increases but inconsistent evidence exists for SS-induced contralateral strength and activation deficits, it is possible, but not clear, that a prior mentally fatiguing task such as prolonged SS at or near the point of discomfort could contribute to impairments.
Nocebo effects	Likely	The limited evidence points to prior knowledge having an effect on subsequent performance.

Δ : change in.

Figure 1: A) The effects of static stretching (SS) with and without a warm-up on performance. **B)** Mechanisms potentially underpinning the SS-induced performance impairment.

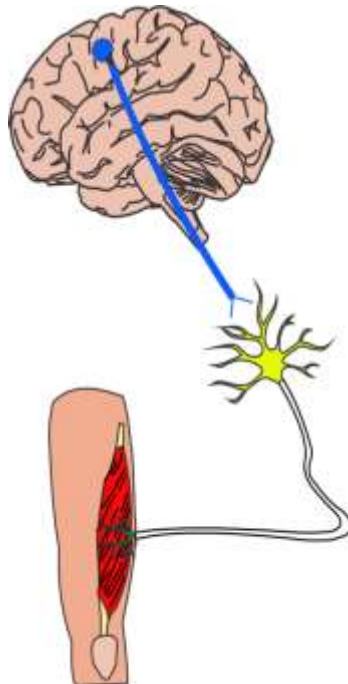
A

Full warm-up with <60 s SS per muscle group		↑ Range of motion ↑ Force at longer muscle lengths
Without warm-up with >60 s SS per muscle group		↑ Range of Motion ↓ Performance (strength, power, endurance, sprint)

B

– Possible or ✓ Likely

- Mental Energy Deficit
- ✓ Nocebo Effects
- ✓ ↓ Persistent inward currents
- ↓ Exteroceptive reflex
- ↓ Parallel elastic (muscle)



✗ Unlikely or ? Unclear

- ✗ Corticospinal excitability
- ✗ Cortical silent period
- ? Hoffman reflex
- ✗ Golgi tendon organ inhibition
- ✗ Series elastic (tendon) stiffness