THE EFFECTS OF ANTAGONIST STRETCHING ON AGONIST PERFORMANCE

By

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Abstract

Introduction The idea of stretching the antagonist muscle to enhance the performance of the agonist muscle has gained considerable attention in recent years. However, most studies have focused on a single stretching duration, leaving a gap in research that compares the effects of varying stretching durations on agonist muscle performance.

Purpose The aim of this study was to compare different durations (40-, 80-, and 120-seconds as well as a control condition) of antagonist static stretching (SS) on agonist muscle performance.

Methods In this randomized crossover study, 16 participants (six females) who do strength training sessions 2-3 times per week regularly were recruited. The study aimed to investigate the acute effects of tibialis anterior (TA) SS with different durations on the plantar flexion (PF) performance and soleus activation. The participants underwent four sessions, with 24-72 hours interval between sessions. Each session included testing pre- and post-SS and one session for control condition without SS. Measurements included the dominant leg's slow (60 degrees/s) and fast (240 degrees/s) PF isokinetic peak torque, isometric peak torque, total work, stiff leg drop jump (SDJ) performance including height, reactive strength index (RSI) and peak power, and finally, electromyography (EMG) from TA and soleus. TA SS protocol involved 1x40 (40s), 2x40 (80s), and 3x40s (120s) with 15-seconds rest in between sets.

Results There were no significant effects of specific antagonist stretching durations on changes in pre- to post-intervention isometric peak torque, slow or fast isokinetic peak torque, total work, or SDJ height, RSI, and peak power ($p > 0.05$). However, when

combining all stretch durations and the control condition (main effect for time), there was a general decrease in fast and slow isokinetic peak torque, total work, and all SDJ measures ($p < 0.05$). Soleus EMG decreased from pre- to post-SS significantly after 120s in both isometric ($p = 0.002$) and slow isokinetic ($p = 0.002$) peak torque as well as 80s in slow isokinetic peak torque ($p = 0.02$).

Conclusions The main finding of this study was different durations of antagonist stretching (40s, 80s, 120s) did not significantly influence the agonist muscles performance. However, when all stretch durations and control conditions were combined, a general decrease was observed in fast and slow isokinetic peak torque, total work, and all SDJ measures, indicating possible testing effects.

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List of Abbreviations

Ca^{2+} – Calcium

EMG – Electromyography

H-reflex – Hoffman Reflex

Hz – Hertz

MTU – Musculotendinous Unit

MVC – Maximal Voluntary Contraction

M-Wave – Compound Muscle Action Potential

PEC – Parallel Elastic Components

PF – Plantar Flexion

PNF – Proprioceptive Neuromuscular Facilitation

POD – Point of Discomfort

ROM – Range of Motion

RSI – Reactive Strength Index

S – Seconds

SDJ – Stiff Leg Drop Jump

SS – Static Stretching

TA – Tibialis Anterior

VJ – Vertical Jumping

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Chapter 1: Literature Review

1.1 Introduction

Stretch training is a popular form of exercise that has been shown to enhance flexibility and joint range of motion (ROM) (Bandy & Irion, 1994; Paradisis et al., 2014), prevent muscle (Takeuchi et al. 2024) and musculotendinous injuries (Amako et al., 2003; Behm et al., 2016a; Behm et al., 2021b), accelerate the recovery and rehabilitation process (Weng et al., 2009), and in some circumstances (e.g., dynamic stretching), improve physical performance (Behm et al., 2016a; Herman & Smith, 2008; Turki-Belkhiria et al., 2014).

There are a variety of stretching methods with different effects and applications. SS involves the elongation of a muscle until a point of initial or maximum pain or discomfort, which is then maintained by holding that position for a prolonged duration (e.g., 15-60 seconds). The duration of the SS can vary according to the aim of the exercise. Shorter durations (<60-s per muscle group) are recommended when SS is incorporated as part of a dynamic warm-up prior to activity (Behm, 2018; Behm et al., 2016a; Behm & Chaouachi, 2011; Behm et al., 2021c; Kay & Blazevich, 2012), whereas when SS is used as a separate training session to increase joint ROM, 30-60 s is recommended as the optimal duration (Behm, 2018). This type of stretching can be categorized as active or passive, where active means the stretch is performed by contracting other muscles, and passive means utilizing an external force such as a resistance band or a person to perform the stretch on a relaxed muscle (Behm, 2018).

Actively moving the limb through the ROM of the related joint refers to dynamic stretching (Behm, 2018). Dynamic stretching is performed in a controlled manner in contrast to another form of dynamic stretching; ballistic stretching which involves more explosive forces (Behm, 2018). Alternatively, performing the movement too slowly may put excessive pressure on the muscle because of higher time under tension, leading to difficulty performing the remaining training volume (Pearson et al., 2022).

Ballistic stretching as stated above involves moving the limb rapidly in a cyclic manner (Bacurau et al., 2009). Finally, proprioceptive neuromuscular facilitation (PNF) is a stretching technique that combines the SS and isometric contraction of the same muscle or group of muscles (Sharman et al., 2006). There are two types of PNF stretching. The "contract relax" method involves passive SS, and then an isometric contraction of the stretched muscle (Sharman et al., 2006). "Contract relax agonist contract" is another type of PNF that is similar to contract relax but with an additional isometric contraction of the antagonist muscle which stretches the agonist muscle (Sharman et al., 2006).

1.1.1 Static Stretching and Physical Performance

The idea of using SS as a physical performance booster began during the World Wars when the general belief was performing SS can increase ROM and flexibility which would lead to improved performance (Behm, 2018), however, studies conducted in the early 2000s started to show the negative effects of an acute bout of prolonged SS on performance (Behm et al., 2001; Cornwell et al., 2002; Fowles et al., 2000; Shrier, 2004). Kay and Blazevich (2012) in a systematic review categorized SS durations into four groups; <30 s, 30-45 s, 1-2 min, and >2 min. They reported that studies with SS duration of <45 s were unable to demonstrate any detrimental effect on performance. Although

significant reductions in performance parameters emerged after performing SS >60 s the chance of significant losses was not further increased by longer durations (>2 min). Similarly, a number of reviews (Behm et al., 2016a; Behm et al., 2021c; Chaabene et al., 2019) highlighted that performing SS <60 s had a trivial negative effect on performance, whereas going beyond 60 s reduced performance significantly. Therefore, SS must be considered from a dose-response point of view.

Chaabene et al. (2019) discussed the potential effects of SS as a part of a warm-up routine, not as a single-mode intervention. It has been suggested that SS ≤60 s in addition to aerobic exercises, dynamic stretching, and sport-specific exercises as a warm-up routine has trivial or positive effects on performance (Behm et al., 2021c; Chaabene et al., 2019). Stretching can also provide the athlete with a more positive psychological view, so that they feel more confident in their subsequent performance (Blazevich et al., 2018; Chaabene et al., 2019). Since prolonged SS may slightly impair the performance, extra care should be taken when performing it prior to high performance sports (Chaabene et al., 2019). Alternatively, a review by Warneke and Lohmann (2024) indicates that although SS in isolation may decrease strength in controlled lab environments, it appears to have no negative impact on athletic performance, such as jumping and sprinting with a full dynamic warm up.

The physiological mechanisms that lead to performance impairments following prolonged SS can be divided into neural, morphological, and psychological (Behm et al., 2021c; Chaabene et al., 2019). Studies that applied SS >60 s revealed a decrease in motor unit activation as evidenced by EMG amplitude reduction (Babault et al., 2010; Ryan et al., 2014; Trajano et al., 2013). Some studies concluded that no EMG changes occur after

a bout of SS (Caldwell et al., 2019; Kay & Blazevich, 2009a; Palmer et al., 2019). However these studies differed as to whether the stretched muscle was not an agonist (Caldwell et al., 2019), the stretching duration was ≤60 s (Palmer et al., 2019), or the pre-SS EMG was not measured immediately post-SS as compared to 30 minutes post-SS (Kay & Blazevich, 2009a). Since EMG-force relationship is a curvilinear slope (Solomonow et al., 1990), reduced motoneuron activity following SS might be a reasonable explanation for muscle force impairments. But, Behm et al. (2021c) discuss that this curvilinear relationship does not necessarily result in equivalent modifications in muscular force. Since EMG signals can experience a plateau while performing maximum or submaximum force, insignificant changes might not be observed in these conditions. Hence it is suggested to normalize the EMG results to M-wave in order to recognize the variations (Behm et al., 2021c; Pulverenti et al., 2020).

Additionally, some studies examined motoneuron excitability by examining the Hoffman (H)-reflex variation after SS. H-reflex is the action potential of a muscle in response to an external stimulation to the sensory nerve representing the afferent excitability of the motoneuron (Palmieri et al., 2004). Theoretically, if SS can affect the H-reflex, then it might be able to affect the afferent neural drive to the α-motoneurons of the related muscle and consequently the muscle performance. Budini et al. (2018) discuss the underlying reasons behind reduced motoneuron excitability highlighting two primary mechanisms: pre-synaptic and post-synaptic inhibition. Pre-synaptic inhibition pertains to the release of inhibitory neurotransmitters or a reduction in the release of excitatory neurotransmitters, which can impact Ca^{2+} channels (Avela et al., 1999). Conversely, post-synaptic inhibition refers to alterations in the post-synaptic membrane potential resulting

from neurotransmitter release by post-synaptic neurons as noted by Avela et al. (1999). Studies show that SS can diminish the amplitude of the H-reflex, however the H-reflex is recovered as soon as the muscle returns to its resting position (Avela et al., 1999; Budini et al., 2018; Stevanovic et al., 2019). This H-reflex recovery can be either fast, due to the pre-synaptic inhibition relief that occurs when the stretched muscle directly returns to the resting state, or slow, which is related to the post-activation depression experienced when the joint transitions from the agonist stretch position to the antagonist stretch state before returning to the resting angle (Budini et al., 2018).

The sensory neurons responsible for innervating both spindle and joint receptors follow a trajectory to reach the spinal cord and somatosensory cortex. This prompts an inquiry into the potential role of altered corticospinal excitability as a potential contributor to decreased force generation following a bout of SS. Chaabene et al. (2019) and Pulverenti et al. (2019) used transcranial magnetic stimulation, a technique to measure corticospinal excitability, to examine motor evoked potential of the muscle in resting state, but they concluded no changes in motor evoked potential amplitude although there were force and EMG reductions following SS. Nonetheless, this does not negate the impact of SS on corticospinal excitability, as the decline in EMG activity could potentially be attributed to a decrease in spinal excitability (Pulverenti et al., 2019).

On the other hand, longer-duration SS causes decrements in the musculotendinous unit (MTU) and muscle's parallel elastic components (PEC) stiffness and increments in MTU compliance that may have an adverse effect on muscle capacity to generate force (Behm et al., 2021c; Chaabene et al., 2019). Behm et al. (2021c) state *"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 fiber forces that is axial (longitudinal) to the line of action of the muscle, and (iii) reducing fiber/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 fibers". It should be noted that MTU compliance can alter the muscle length-tension relationship which can move the curve to the right. (Kallerud & Gleeson, 2013). Therefore, the stretched muscle will not be in optimal length for generating force when it is returned to its original joint angle that leads to a decrease in maximal voluntary contraction (MVC) (Fowles et al., 2000). However, it can result in higher force production at longer muscle lengths, where most muscle strain injuries occur (Behm et al. 2021).

In addition, prolonged activities put the person in the danger of perception of fatigue even if the activity is not actually fatiguing (Behm et al., 2021a). Steele (2020) define the perception of fatigue as *"…the perception of that which must be done in attempting to achieve a particular demand, or set of demands, and which is determined by the perception of current task demands relative to the perception of capacity to meet those demands…"*. Gandevia (2001) introduced the psychological concept termed the 'Global Sensory Tolerance Limit' to elucidate the perception of fatigue. In addition, Behm et al. (2021a) proposed that the cumulative negative sensory feedback from muscles, originating directly from targeted muscles or indirectly from core stabilizers and other fatigued non-local muscles, could collectively exert a detrimental influence on subsequent performance. Therefore, SS for prolonged periods of time gives the person the perception that the next

activity will be more difficult, and as a result, the person may stop the next task prematurely or not be able to perform to their full potential (Behm et al., 2021c; Pageaux et al., 2014).

Finally, there are not many studies explaining placebo or nocebo effects of SS, however, there were trivial performance improvements such as increase in total volume and total number of repetitions after 90 seconds of SS when participants were biased positively (Bertolaccini et al., 2021; Janes et al., 2016). These participants were provided with positive information (orally, visually, and in a written manner) that SS improves performance.

1.1.2 Static Stretching and Range of Motion

SS has been utilized for many years as a method to improve flexibility and increase joint ROM (Ayala et al., 2013; Kataura et al., 2017; Medeiros et al., 2016; O'Sullivan et al., 2009). Increased flexibility helps the individual to use the limb through a greater ROM, and therefore an improvement in performance and activity (Bryant et al., 2023). On the other hand, lower flexibility, which is linked to higher MTU stiffness (Watsford et al., 2010; Witvrouw et al., 2003), might increase the risk of injury since the need for energy surpasses the MTU's stiffness capacity (Bryant et al., 2023; Lorimer & Hume, 2016).

There are certain variables that may be managed and taken into account while using SS as flexibility training. The duration or volume of SS is the first factor. The results regarding the impact of SS duration on ROM are inconclusive. When the total duration is equal (3 sets of 15 s vs. 9 sets of 5 s; total 45 s), a study found no difference in ROM improvement across groups (Roberts & Wilson, 1999). However, when comparing 30 s,

60 s, and 120 s of SS, no significant difference between groups was found (Palmer et al., 2019). Furthermore, Donti et al. (2014) evaluated 15 s and 30 s of SS and discovered that 30 s of SS can considerably enhance ROM, but 15 s of SS had no discernible effects. On the other hand, a few studies revealed a significant increase in ROM even after 15 s of SS. (Bandy & Irion, 1994; Tsolakis & Bogdanis, 2012). Another study by Kurtdere et al. (2020) concluded that there is no difference in ROM improvements when the participants do higher durations of SS (3.5 vs. 7 vs. 10.5 minutes). It appears that performing SS for longer than a specific duration has no further beneficial effects on joint ROM. Contrarily, shorter SS sessions (100 and 240 seconds) increased hamstrings flexibility significantly more than longer sessions (240 and 900 seconds), but the intensity of the shorter sessions was also significantly higher (100 and 120% vs. 50%) (Freitas et al., 2016; T. Fukaya et al., 2020). In summary, Behm et al. (2023) suggest that ROM can be improved with only 5 s of SS, while 240 s is the greatest, based on comparisons of duration dependent research conducted without taking into account the intensity of stretching variations. Therefore, the intensity of stretching is yet another component that is crucial.

The concept of SS intensity has been defined in various ways within the literature. McClure et al. (1994) initially introduced one definition, describing it as the level of force applied to a joint during stretching, which may be limited by an individual's pain tolerance. Another perspective from Freitas et al. (2015) characterizes it as *"…the degree of muscle tendon lengthening induced by a change in joint range of motion (ROM), that is controlled by subjective assessment of human tolerance to stretch using the criteria of pain or discomfort."*. In practice, different approaches are employed to gauge SS intensity. Some researchers gauge it as a percentage of the point of discomfort (POD) (Behm & Kibele,

2007; Taizan Fukaya et al., 2020; Young et al., 2006). Meanwhile, Freitas et al. (2016; 2015) calculated SS intensity as a percentage of the maximum-tolerated passive joint torque.

One study investigated SS intensities at 80%, 100%, and 120% of the maximum tolerable intensity without inducing stretching pain. It concluded that the 120% intensity led to the most significant increase in ROM, while no significant improvement in flexibility was observed with 80% intensity (Kataura et al., 2017). Additionally, Takeuchi and Nakamura (2020) reported that stretching at the maximum POD, can enhance flexibility more than using 120% or 100% POD. It's important to highlight that in this study, POD is defined as the moment just before participants begin to sense a mild discomfort (Takeuchi & Nakamura, 2020). Multiple studies have consistently demonstrated that higher SS intensities result in greater ROM increases (Freitas et al., 2016; Freitas, Vilarinho, et al., 2015; T. Fukaya et al., 2020; Fukaya et al., 2021; Nakamura et al., 2020; Nakamura et al., 2022). However, the effect of SS intensity on ROM gains is inconclusive, with no significant modulation observed between high and low intensities (Behm et al., 2023). This inconsistency arises partly from varied definitions of intensity, such as discomfort, pain threshold, or maximum ROM with mechanical assistance (Behm et al., 2023).

In contrast, Santos et al. (2020) did not observe any significant differences in ROM improvements when comparing low and high SS intensities. Two other studies supported this finding, although one of them had a longer duration for the lower intensity group (Marchetti et al., 2022), potentially influencing the results, and the other examined changes after 10 sessions of SS training rather than the acute effects (Melo et al., 2021). Also, Konrad et al. (2024) reviewed the chronic effects of SS training on ROM and found that

both high and low intensities have moderate effects on ROM, with low intensity stretching being nearly as effective as high-intensity. However, these conclusions primarily apply to recreationally active or trained individuals, and additional research is needed for those requiring extreme flexibility, such as gymnasts and figure skaters (Konrad et al., 2024)

Several theories exist to elucidate the mechanisms underpinning enhanced flexibility following a session of SS. One such theory pertains to stretch tolerance, wherein, post-SS, an individual can achieve a greater ROM while perceiving the same level of stretching intensity. (Ben & Harvey, 2010; Blazevich et al., 2014; Brusco et al., 2019; Bryant et al., 2023). Another explanation revolves around alterations in mechanical properties. Kay and Blazevich (2009b) documented noteworthy reductions in muscle stiffness following a 180 s SS session. Furthermore, Nakamura et al. (2011) reported significant decreases in both muscle stiffness and the MTU following 5 min of SS. In sum, SS proves to be an effective method for augmenting flexibility, with its outcomes influenced by factors like duration and intensity.

1.2 Non-local Stretching Effects

Lately, there has been a growing body of research indicating the presence of non-local or global stretching effects (Behm et al., 2016b; Caldwell et al., 2019; De-la-Cruz-Torres et al., 2021; Killen et al., 2019). Non-local or crossover effects of stretching occur when stretching a specific muscle or group of muscles results in changes in the ROM or performance of either a homologous (crossover) or heterologous (non-local) muscle that has not been stretched (Behm et al., 2021d; Caldwell et al., 2019). Notably, Clark et al. (1999) were the first to discover that stretching the left hamstrings positively impacted the flexibility of the right hamstrings. Furthermore, Behm et al. (2016b) observed that

stretching the shoulder (specifically, shoulder horizontal abduction) led to increased passive hip flexion ROM with the hamstrings, while stretching the lower body (hip adductors) improved shoulder ROM (shoulder extension). Finally, Behm et al. (2021d) conducted a systematic review on this topic and concluded that unilateral stretching improves ROM to a "moderate" magnitude extent.

Since the homologous or heterologous limb is not subjected to any stretching, there can be no mechanical explanation for the non-local stretching effect (Chaouachi et al., 2017). Therefore, it is plausible to consider neural factors in this context. The reduction in motoneuronal excitability, as indicated by a decrease in EMG and H-reflex activity after prolonged SS (Anvar et al., 2023; Behm et al., 2001; Behm et al., 2013) is likely due to a decrease in sensory input from muscle spindles, specifically type I and type II afferents (Guissard et al., 1988). In addition, Amann et al. (2013) propose that type III and type IV afferents may send inhibitory signals to the corticospinal pathways. These combined effects lead to a diminished central drive to the muscles, ultimately allowing contralateral or other muscles to achieve a greater ROM (Amann et al., 2013; Behm et al., 2016b; Chaouachi et al., 2017). However, Chaouchai et al. (2017) discovered that there were no EMG alterations in the contralateral limb following 240 s of unilateral SS, casting uncertainty on the notion of reflex-driven reductions in motoneuron excitability. Finally, the most robust explanation for non-local stretching effects is an enhanced capacity to tolerate stretching. Magnusson et al. (1996a; 1996b) observed an increase in passive peak torque induced by stretching without significant alterations in EMG activity. This suggests that due to the extensive interconnection of cortical structures, an improved tolerance for stretching would likely affect the entire body.

When considering the impact of non-local stretching effects on performance, there is limited research available. However, some studies have shown significant effects. For example, Marchetti et al. (2014) found that performing 10 sets of 30 seconds of shoulder SS can notably decrease the ground reaction force in countermovement jumps. Additionally, Cè et al. (2020) observed that 5 sets of 45 seconds of unilateral quadriceps SS led to a substantial reduction in MVC of the contralateral knee extensors. On the other hand, other studies have reported either no change (Chaouachi et al., 2017; Jelmini et al., 2018) or trivial impairment (Caldwell et al., 2019; Killen et al., 2019) in contralateral limb performance following prolonged SS. Finally, a systematic review conducted by Behm et al. (2021e) concluded that non-local SS typically results in "small magnitude" performance impairments.

1.3 Antagonist Muscle Stretching Effects on Agonist Muscle Performance

Several investigations have explored how the use of antagonist SS affects the performance of agonist muscles. These studies have employed varying SS durations and protocols. For instance, one study revealed a significant increase in maximum power during the bench press when participants underwent 30s of SS targeting shoulder adductors and protractors (Elliott & Massey, 2020). Additionally, performing 3 sets of 30 s (90 s) of SS on hip flexors and dorsiflexors, which are antagonist muscles during vertical jumping (VJ), led to increased VJ height (Sandberg et al., 2012; Wakefield & Cottrell, 2015), VJ power (Sandberg et al., 2012), and peak isokinetic torque at higher speeds ($300°.s^{-1}$) (Sandberg et al., 2012). Another study employed the same duration of SS to stretch knee flexors, another antagonist for VJ, and observed a significant improvement in VJ height (Mendes Leal de Souza et al., 2016). Although Jones & Humphrey (2018) reported

improvements in VJ height, VJ power, and knee extensors peak power following the same duration of SS, the differences were not substantial.

In contrast, some studies used a single set of 40 s SS for antagonist muscles between sets of seated row exercises and found a significant increase in total training volume (the number of sets multiplied by the number of repetitions multiplied by the load) and activation of agonist muscles. However, they did not observe any changes in antagonist muscle activation (Miranda et al., 2015; Paz et al., 2016; Paz et al., 2013). On the other hand, a study using the same duration of SS on knee flexors but in a different manner (2 sets of 20 s) did not find any changes in knee extension total training volume (Pessoa et al., 2023).

Some studies employed longer durations of SS. For example, 4 sets of 30 s (120 s) SS on VJ antagonists increase VJ height meaningfully (Sekir et al., 2016). Cogley et al. (2021) investigated the effects of 8 sets of 30 s (240 s) SS on knee flexors on fast ($300°.s^{-1}$) and slow ($60°.s^{-1}$) knee extension for average power and found only a significant increase in the faster speed. However, Serefoglu et al. (2017) used the same SS protocol and did not find any significant increases in either slower ($60°.s^{-1}$) or faster ($240°.s^{-1}$) knee extension torque, although there were slight improvements. Also, two studies utilizing 5 sets of 45 s (225 s) (Cè et al., 2021) and 5 sets of 60 s (300 s) (Montalvo, 2021) SS of antagonist muscles showed no substantial improvement in agonist MVC and VJ height, respectively.

In summary, the majority of studies examining the effects of antagonist SS on agonist performance reported significant performance enhancements regardless of their SS duration. Only 2 studies failed to identify any improvements in muscular performance. Therefore, it can be concluded that antagonist SS can have a positive impact on agonist

muscle performance. However, all these research studies focused solely on specific durations of SS. To date, no study has explored and compared how varying durations of SS might influence the extent of performance enhancement or what the optimal duration of antagonist SS is for improving agonist performance.

1.3.1 Antagonist Static Stretching Mechanisms

During a movement, both the agonist and antagonist muscles are activated simultaneously (Tillin et al., 2011). This simultaneous contraction and activation of the agonist and antagonist is commonly referred to as "coactivation" or "co-contraction" (Folland & Williams, 2007) which can hinder the agonist from achieving its full force or power output. This phenomenon is known as the "braking effect" (Dal Maso et al., 2012) and Golgi tendon organs of the agonist and muscle spindles in the antagonist contribute to this effect (Sharman et al., 2006). The force produced during a motion is directly influenced by the force of the agonist muscle and is inversely affected by the force of the antagonist muscle (Baratta et al., 1988; Draganich et al., 1989). Hence, enhancing the force of the agonist muscle can be achieved through two avenues: by improving the force it generates itself or by reducing the force generated by the antagonist. Applying SS to the antagonist which has been shown to inhibit neural drive to the muscle (Fowles et al., 2000; Robbins et al., 2010) is a way to decrease its force generation and coactivation. Ultimately, this can lead to an improvement in the force generated by the agonist.

A possible explanation for the reduction in coactivation might be lower motor unit activation of the antagonist following SS. Previous studies examining agonist SS effects on its performance have shown that applying SS >60 s to the agonist decreases its EMG amplitude (Babault et al., 2010; Ryan et al., 2014; Trajano et al., 2013), and it is suggested

to normalize the EMG to the M-wave to get more accurate and reliable results (Behm et al., 2021c; Pulverenti et al., 2020). However, the findings of antagonist SS are contradictory. These studies used 40 s (Miranda et al., 2015; Paz et al., 2016; Paz et al., 2013), 90 s (Sandberg et al., 2012), 225 s (Cè et al., 2021), and 300 s (Montalvo, 2021) antagonist SS and none of them could find a significant decrease in antagonist EMG amplitude. However, when Montalvo (2021) divided the participants into trained and untrained groups, they found that only the untrained group illustrated a significant decrease in antagonist EMG. Among these studies that measured antagonist SS, only Cè et al. (2021) normalized the results to the M-wave and were unable to show significant differences either. Although more studies need to be performed considering participants' training status, it can be concluded that antagonist SS regardless of its duration does not affect its motor unit activation level.

These studies looked at the EMG variations of the agonist following antagonist SS as well. Sandberg et al. (2012) found that 90 s knee flexors SS was unable to increase knee extensors EMG significantly. SS of dorsiflexors and hip flexors for 120 s could not affect the EMG activity of plantar flexors and hip extensors either, respectively (Sekir et al., 2016). Also, 240 s and 225 s of knee flexors and plantar flexors failed to make any meaningful changes to knee extensors and TA EMG activity, respectively (Cè et al., 2021; Serefoglu et al., 2017). It should be noted that surface EMG might not be sensitive enough to recognize notable changes (Miranda et al., 2015; Paz et al., 2013). On the other hand, only 40 s SS of pectoralis major and shoulder adductors could increase latissimus dorsi and biceps brachii EMG activity substantially (Miranda et al., 2015; Paz et al., 2016; Paz et al., 2013). It is really important to note that all of the studies that illustrated EMG increment

showed performance improvement too. Therefore, one potential explanation for agonist performance improvement following antagonist SS might be an increase in neural drive and motor unit activation of the agonist. In conclusion, since only 40 s SS on a specific muscle group has shown agonist EMG increment following antagonist SS, these results might be muscle-, exercise-, and/or SS duration-specific, however, more studies should be done on different muscle groups and durations of SS.

Another possible interpretation involves either a decrease in the motoneuron excitability of the antagonist or an increase in the agonist motoneuron excitability. Previous research investigating the impact of SS on H-reflex amplitude has indicated that SS can temporarily decrease H-reflex amplitude during the stretching period, but it typically returns to its original level once the muscle returns to a relaxed state (Budini et al., 2018; Masugi et al., 2017; Stevanovic et al., 2019). However, in the context of stretching the antagonist muscle, only two studies have explored variations in H-reflex, yielding conflicting outcomes. Masugi et al. (2017) observed that 30 s SS applied to the plantar flexors not only decreased spinal reflex excitability in the soleus and gastrocnemius but also in the TA. Nevertheless, this reduction was observed only during the stretching period and reverted to the baseline once stretching was concluded. The explanation provided was based on the non-local effects of SS and reciprocal inhibition triggered by signals from the stretched muscles (specifically Ia afferents through Ia interneurons) (Masugi et al., 2017). On the other hand, another study normalized the H-reflex to the M-wave and found no alterations in the H/M ratio of the TA following plantar flexors SS for 5 sets of 45 s, indicating no changes in spinal reflex excitability and neuromuscular function (Cè et al., 2021).

Sandberg et al. (2012) suggest that antagonist SS changes MTU compliance which leads to a change and disruption in the muscle length-tension relationship (Fowles et al., 2000; Kallerud & Gleeson, 2013). This change decreases the magnitude of the braking effect and might cause an increase in agonist performance (Sandberg et al., 2012). Also, Miranda et al. (2015) found that the 12-15% increase in repetition performance after antagonist SS was similar to the 12-21% decrease in repetition after agonist SS examined in another study (Gomes et al., 2011). However, the author's conclusion is questionable, because these two studies used different duration of SS and different exercises.

Studies investigating the effects of antagonist SS on higher-speed peak torque or power (Cogley et al., 2021; Elliott & Massey, 2020; Sandberg et al., 2012) have shown significant improvements. Some studies have also observed slight improvements in power or fast development of peak torque (Jones & Humphrey, 2018; Serefoglu et al., 2017). On the other hand, studies measuring slower peak torque in knee extension found only slight and non-significant increases following antagonist SS (Cogley et al., 2021; Montalvo, 2021; Sandberg et al., 2012; Serefoglu et al., 2017). Sandberg et al. (2012) have suggested that the positive effects of antagonist SS might be specific to velocity and primarily occur during faster movements. However, further research is required to confirm this hypothesis. It is worth noting that the duration of SS sessions in these studies varies, and some of them used a velocity of $240°.s^{-1}$ while others considered $300°.s^{-1}$ as faster velocity. To obtain more precise results, it's essential to control these variables. It's important to highlight studies using a velocity of $300°.s^{-1}$ for assessing average power and peak torque have consistently found significant positive results (Cogley et al., 2021; Sandberg et al., 2012), while a velocity of $240°.s^{-1}$ did not yield significant improvements (Serefoglu et al., 2017).

Consequently, the beneficial effects of antagonist SS may be specific to a velocity of 300°.s^{-1} or even higher.

Numerous studies have highlighted the potential for substantial performance loss after SS sessions of the target (agonist) muscle lasting more than 60s (Behm et al., 2016a; Behm et al., 2021c; Chaabene et al., 2019). This implies that significant performance improvements in agonist muscles might occur when stretching the antagonist for durations exceeding 60s. However, it's important to note that some studies have reported significant performance enhancements even with shorter durations of antagonist SS, such as 40 s (Miranda et al., 2015; Paz et al., 2016; Paz et al., 2013) or 30 s (Elliott & Massey, 2020).

In summary, the primary factor contributing to the enhanced performance of the agonist muscle following antagonist muscle SS is the decrease in force generation by the antagonist. This reduction in antagonist force decreases the resistance to the agonist muscle force output. However, it is worth noting that not all studies demonstrate improved neuromuscular function in the agonist muscle, indicating that this may not be the predominant factor behind performance improvement following antagonist SS.

1.4 Conclusion

The concept of antagonist SS to enhance the performance of the agonist muscle has gained popularity, and previous studies have demonstrated its positive effects (Elliott & Massey, 2020; Mendes Leal de Souza et al., 2016; Paz et al., 2013; Sandberg et al., 2012). All the studies that looked at this topic have used only one SS protocol to observe the effects. However, existing studies on this subject have typically utilized a single SS

protocol to assess its impact. Notably, there is a lack of research comparing the effects of different durations of antagonist SS on agonist muscle performance.

1.5 Research Objectives

This study sought to analyze the impact of varying durations of antagonist (tibialis anterior) SS on the performance of agonist (plantar flexors) muscles. The study specifically compared the effects of three different durations of SS on the antagonist muscles: 40 s (1 x 40), 80 s (2 x 40), and 120 s (3 x 40). The performance of two movements, namely ankle PF isokinetic and isokinetic peak torque and SDJ were evaluated.

1.6 Hypotheses

Based on the previous research both 40 s and 120 s SS of antagonist muscles have a positive significant effect on agonist performance (Miranda et al., 2015; Paz et al., 2016; Sandberg et al., 2012). Also, it has been shown that 90 s of antagonist SS leads to significant improvement in agonist performance (Wakefield & Cottrell, 2015). Hence, it was hypothesized that all the proposed SS durations would improve agonist performance. However, as of today the main reason for this improvement has shown to be antagonist muscle force reduction and decrease in the rate of coactivation. Therefore, it was hypothesized that 120 s of antagonist SS would cause the most significant force reduction of the antagonist muscle and would positively impact the agonist muscle more than the shorter SS durations.

Chapter 2: Research

2.1 Methods

2.1.1 Participants

An “a priori” statistical power analysis (software package, G * Power 3.1.9.7) was conducted based on effects of different SS durations on performance-related studies (Franco et al., 2008) to achieve an alpha of 0.05, an effect size of 0.5, a statistical power of 0.8, and a correlation of 0.5 using the F-test family. The analysis indicated that 16 participants should be sufficient to achieve adequate statistical power. Sixteen (16) healthy active participants took part voluntarily in this study (Table 1). Exclusion criteria included participants with a current injury to the quadriceps, hamstrings, or calf muscles and any injury in hip, ankle, or knee joints, medical issues that prevent performing a high-intensity exercise, or neurological conditions. Inclusion criteria included that participants need to be healthy, between 18-40 years old, and engage in strength training sessions 2-3 times per week on a regular basis.

Table 1: Participant characteristics

Participants	Age (years)	Mass (kg)	Height (cm)
Male (n=10)	30.5 ± 3.77	84.93 ± 14.48	176.96 ± 3.54
Female (n=6)	27.67 ± 3.09	59.47 ± 5.5	161.33 ± 6.21

Prior to their lab visit, participants were given instructions to avoid intense activity (24 hours prior to participating) and to stop drinking alcohol, smoking, and using caffeine (12 hours). Each participant completed the Physical Activity Readiness Questionnaire plus

(PAR-Q+ 2022), read and signed the informed consent form prior to testing and after a brief explanation of the study and the experiment's procedures. During their first visit to the lab, every participant became familiar with all measurements. The Interdisciplinary Committee on Ethics in Human Research (ICEHR #20241815-HK) gave its approval for this study, which was carried out in accordance with the most recent version of the Helsinki Declaration

2.1.2 Experimental Design

The effects of different antagonist stretching durations on physical performance were investigated using a randomized crossover study design. The participants became familiar with a basic orientation to the testing procedures and equipment during the initial familiarization session which was on a separate day from the testing sessions. They performed SDJ, slow and fast isokinetic as well as isometric PF peak torque tests pre- and post-SS. The participants then came to the lab for four distinct testing sessions with SS durations of 40s, 80s, 120s, and control. Each session was randomized and separated by 24-72 hours.

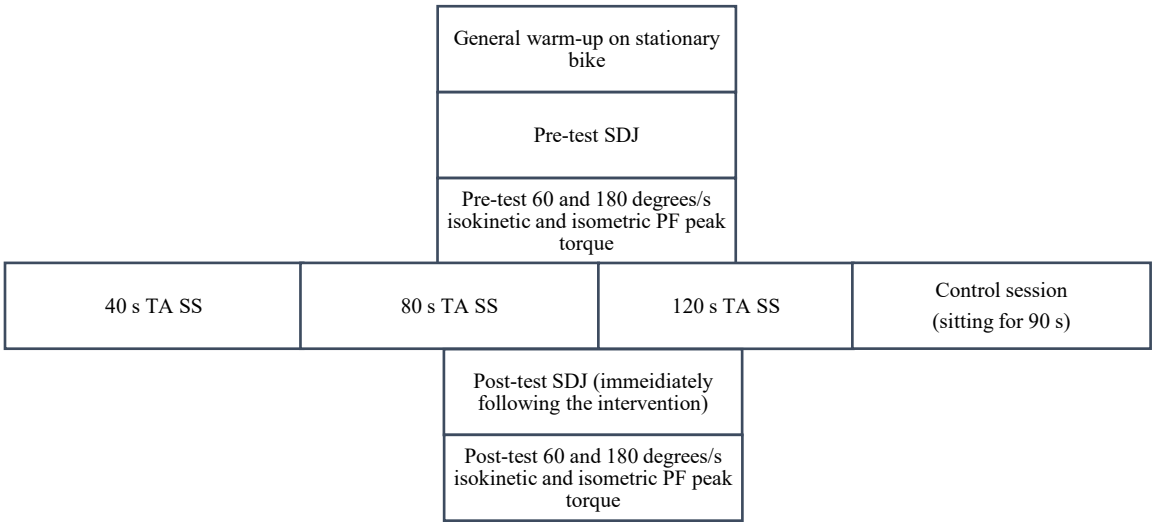

Figure 1 Experimental Design. The interventions were on four different days and the order was randomly assigned.

Acronyms: PF: plantar flexion, SDJ: Stiff leg drop jump, SS: static stretching, TA: tibialis anterior

2.1.3 Independent Variables: Stretch Interventions

To passively stretch the TA, participants lied supine on a padded table with their feet hanging over the edge. An investigator moved both feet into PF by pulling on the distal segment of the foot and stretching the TA until POD. The thighs were strapped tightly to the training bed to minimize knee flexion. Among these sessions, three involved interventions (antagonist SS) of varying durations (40 s, 80 s, and 120 s), and one served as a control (no stretching) session.

2.1.4 Dependent Variables: Measures

2.1.4.1 Electromyography

Surface EMG was employed to document the muscle activity of the dominant soleus and TA. Self-adhesive Cl/AgCl bipolar electrodes (MeditraceTM 130 ECG conductive adhesive electrodes, Syracuse, USA) were utilized in alignment with the muscle fibers and systematically positioned based on the guidelines outlined in "Surface Electromyography for the Non-Invasive Assessment of Muscles" (SENIAM) (Hermens et al., 1999). Prior to placing the electrodes on the skin, the investigators prepared the area by shaving, abrading, and cleaning the skin with an isopropyl alcohol swab, allowing it to dry afterward (Hermens et al., 1999). The ground electrode was positioned on the lateral epicondyle of the femur, and all leads were secured to the skin to minimize potential movement artifacts in the surface EMG signal. Before commencing the experiment, a check was conducted to evaluate the inter-electrode noise, ensuring it remained below five kilo-ohms (5 kΩ). The EMG signals were amplified 1000x (CED 1902 Cambridge Electronic Design Ltd., Cambridge, UK) and filtered with a 3-pole Butterworth filter

having cut-off frequencies of 10-500 Hz. Analog signals were digitally converted at a sampling rate of 5 kHz using a CED 1401 interface (Cambridge Electronic Design Ltd., Cambridge, UK) and sampled at 2000 Hz.

2.1.4.2 Ankle Plantar Flexion Isokinetic and Isometric Peak Torque

Participants were directed to sit on a Humac Norm Isokinetic Machine (Computer Sports Medicine Inc., Stoughton, MA ,USA) chair. Upon being seated, they were secured to the chair using chest straps to minimize extraneous movements during the experiment. The chair back angle was set to 110°. The test was done for the dominant side. To hold the leg, participants' thigh was held and strapped on a thigh stabilizer pad. Participants' dominant knee angle was set to 150°. Then, their dominant foot was placed and strapped on a foot plate. The EMG leads were connected to the electrodes. A goniometer was employed to achieve an ankle angle of 90° for all participants. Three different tests were performed by participants: a slow isokinetic (60 degrees/s), a fast isokinetic (240 degrees/s), and an isometric peak torque. The order of the tests was randomized in each session. The starting point of the isokinetic tests was set to 10° of dorsiflexion and the ending point at 40° of PF. Also, for the isometric test, the ankle angle was set to 10° of dorsiflexion based on manufacturer's instructions. Participants did three maximum isokinetic PF at each speed, and the maximum peak torque among these was chosen. Additionally, participants did 2-3 isometric PF MVCs in 10° of dorsiflexion. The peak torque value obtained during the first MVC was recorded. If the value for the second MVC was 5% greater than the first, a third MVC was conducted to ensure that the participant reached their maximum force production.

2.1.4.3 Stiff Leg Drop Jump (SDJ)

To evaluate the SDJ performance of the participants, the Chronojump Boscosystem Contact Platform Kit (Chronojump-Boscosystem, Australia) was utilized. Previous studies supported the validity and reliability of this system ((Pueo et al., 2020; Villalon et al., 2024). Participants were guided to stand on a step 30 cm above the ground with their hands on their hips (akimbo). They were directed to perform an SDJ where they were instructed to minimize knee and hip flexion while performing the jump to maximize the engagement of plantar flexors. When they jumped, they landed on the platform, and the Chronojump Boscosystem software measured their jump height, jump power, and RSI (jump height / contact time). Participants made three attempts, with a 1-minute rest interval between each and the height one out of three was reported.

2.1.4.4 Statistical Analysis

Statistical analyses were calculated using SPSS software (Version 28.0, SPSS, Inc, Chicago, IL). This study employed a repeated measures, within-subjects, crossover design. Kolmogorov–Smirnov tests of normality were conducted for all dependent variables. Significance was defined as $p \leq 0.05$. The Shapiro-Wilk test indicated all data were normally distributed. Mauchly's test indicated that the assumption of sphericity was met for all measurss (Tables 2, 4, 6, 8). If the assumption of sphericity was violated, the Greenhouse−Geiser correction was employed. Since every subject underwent four sessions (40 s, 80 s, 120 s, and control) and during each session two measurements were done (pre- and post-SS), a 4x2 repeated measures ANOVA was employed. Bonferroni post-hoc tests were conducted to detect significant main effect differences between SS durations whereas, for significant interactions, Bonferroni post-hoc t-tests corrected for multiple comparisons

(α-value divided by the number of analyses on the dependent variable) were conducted to determine differences between values. Partial Eta-squared (η_p^2) values are reported for main effects and overall interactions representing small ($0.01 \leq \eta_p^2 < 0.06$), medium ($0.06 \leq \eta_p^2 < 0.14$) and large ($\eta_p^2 \geq 0.14$) magnitudes of change (Cohen, 2013). Cohen's d effect sizes are reported for the specific post-hoc interactions with d > 0.2: trivial, 0.2 - <0.5: small, 0.5 - <0.8: moderate, ≥0.8: large magnitude difference (Cohen, 2013).

2.2 Results

The Shapiro-Wilk test indicated all data were normally distributed.

2.2.1 Isometric and Isokinetic Peak Torque

There were no statistically significant interaction of duration*time (Figures 2-4), main effect of duration or time (Table 3) for any condition with the exception of a main effect for time for the slow and fast isokinetic peak torque. The main effect of time for fast ($p < 0.0001$, $\eta^2 = 0.684$) and slow ($p = 0.05$, $\eta^2 = 0.230$) isokinetic peak torque decreased significantly from Pre- to Post-test.

Table 2: Table 2 illustrates Mauchley's test of sphericity and significance values for isometric, as well as slow and fast isokinetic peak torque. Shaded boxes highlight significant differences.

	Mauchley's Sphericity assumption met		Significance		
	Duration *time	Duration	Duration *time	Duration Main Effect	Time Main Effect
Isometric Peak Torque	($\chi^2(5)$ = 7.343, p = 0.19)	($\chi^2(5)$ = 0.630, p =0.98),	($F_{(3, 45)}$ = 1.905, p = 0.14, η^2 =0.11)	($F_{(3, 45)}$ = 1.219, p = 0.314, η^2 =0.07)	($F_{(1, 15)}$ = 0.350, p = .56, η^2 =0.02).
Slow Isokinetic Peak Torque	($\chi^2(5)$ = 17.178, p = 0.004)	($\chi^2(5)$ = 4.071, p = 0.54),	F(1.784, 26.753) = 2.040, p = 0.15, η^2 = 0.12	F(3, 45) = 0.824, p = 0.48, η^2 = 0.05	F(1, 15) = 4.478, **p = 0.05,** η^2 = 0.23
Fast Isokinetic Peak Torque	$\chi^2(5)$ = 23.207, p <0.0001	$\chi^2(5)$ = 9.459, p = 0.093	F(1.753, 26.288) = 0.953, p = 0.38, η^2 = 0.06	F(3, 45) = 0.402, p = 0.75, η^2 = 0.02	F(1, 15) = 32.414, **p<0.0001,** η^2 = 0.68

Table 3: Mean and standard deviation (mean±SD) data illustrated for all peak torque measures.

	Isometric Peak Torque	Slow Isokinetic Peak Torque	Fast Isokinetic Peak Torque
Pre40	171.5±43.9	97.5±31.1	57.0±14.4
Post40	175.6±49.9	93.9±31.8	53.2±15.9
Pre80	188.9±48.4	102.6±28.8	60.6±14.5
Post80	183.6±47.8	103.1±30.2	55.1±14.6
Pre120	187.1±42.9	100.6±26.2	57.7±14.2
Post120	188.9±43.7	103.6±29.2	56.1±11.7
Pre-Con	183.1±53.3	102.9±29.9	58.1±11.8
Post-Con	179.1±50.6	97.3±26.5	55.5±14.3

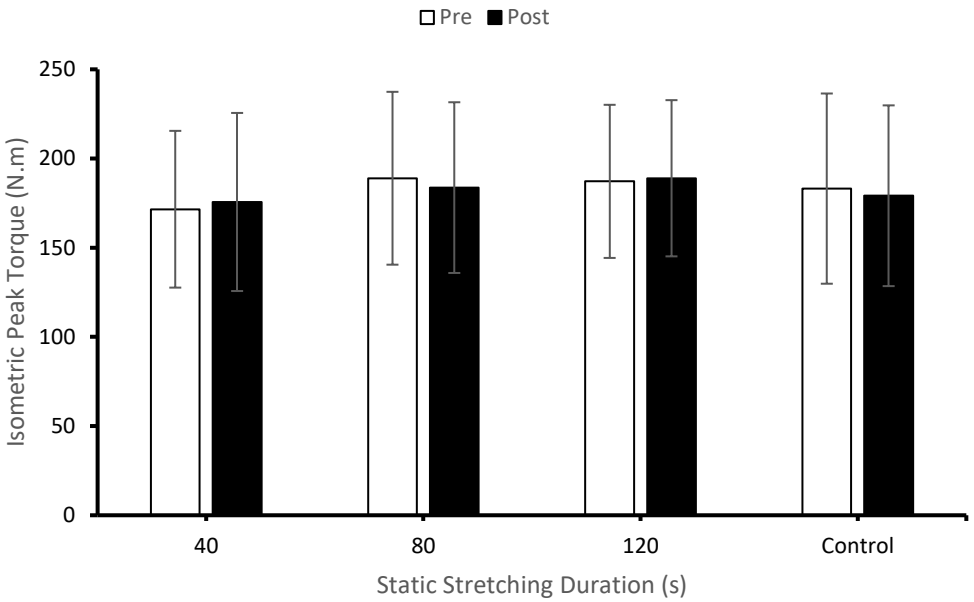

Figure 2: Figure 2 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for isometric peak torque. There were no significant findings.

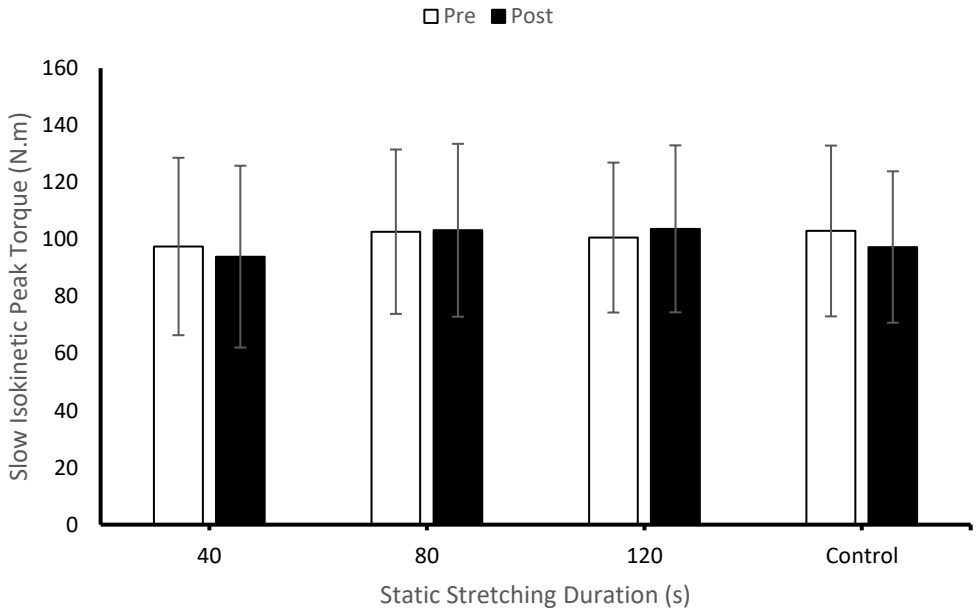

Figure 3: Figure 3 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for slow isokinetic peak torque. There were no significant findings.

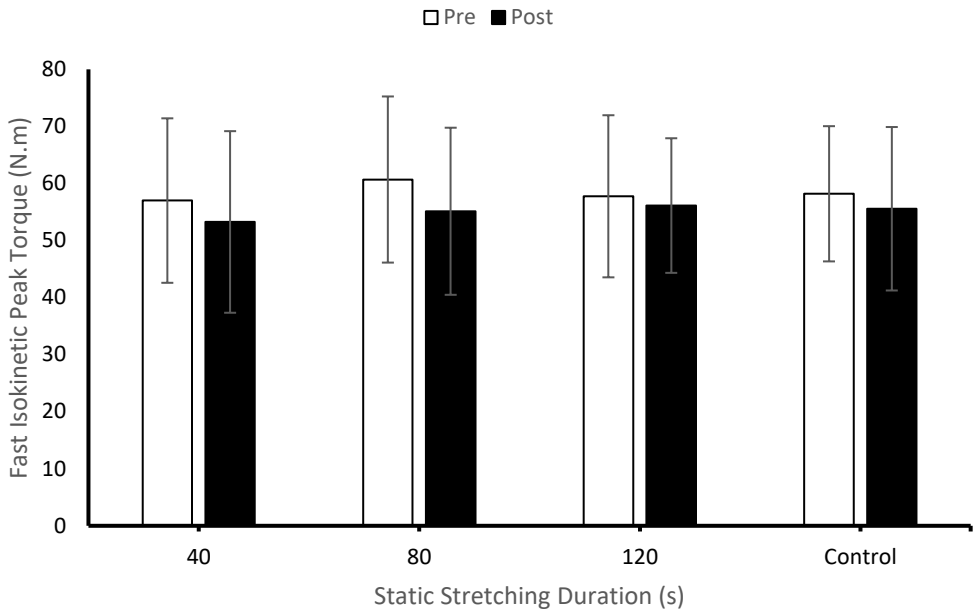

Figure 4: Figure 4 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for fast isokinetic peak torque. There were no significant findings.

2.2.2 Total Work

There were no statistically significant interaction of duration*time (Figures 5, 6), the main effect of duration or time (Table 5) for any condition with the exception of a main effect for time for the fast isokinetic total work. The main effect of time for fast isokinetic total work decreased significantly from Pre- to Post-test ($p = 0.025$, $\eta^2 = 0.294$). A similar decrease in total work was evident with the slow isokinetic contraction, although it did not reach significance ($p=0.06$).

Table 4: Table 4 illustrates Mauchley's test of sphericity and significance values for slow and fast isokinetic total work. Shaded boxes highlight significant differences.

	Mauchley's Sphericity assumption met		Significance		
	Duration *time	Duration	Duration *time	Duration Main Effect	Time Main Effect
Slow Isokinetic Total Work	$\chi^2(5) = 10.957$, $p = 0.05$	$\chi^2(5) = 2.395$, $p = 0.79$	($F_{(3, 45)} = 1.159$, $p = 0.33$, $\eta^2 = 0.07$)	$F_{(3, 45)} = 0.156$, $p = 0.92$, $\eta^2 = 0.01$	$F_{(1, 15)} = 3.905$, $p = 0.06$, $\eta^2 = 0.21$
Fast Isokinetic Total Work	$\chi^2(5) = 1.060$, $p = 0.95$	$\chi^2(5) = 2.112$, $p = 0.65$	$F_{(3, 45)} = 1.400$, $p = 0.255$, $\eta^2 = 0.085$	$F_{(3, 45)} = 0.708$, $p = 0.55$, $\eta^2 = 0.04$	$F_{(1, 15)} = 6.242$, $p = 0.02$, $\eta^2 = 0.29$

Table 5: Mean and standard deviation (mean±SD) data illustrated for all total work measures

	Slow Isokinetic Total Work	Fast Isokinetic Total Work
Pre40	159.7±51.3	81.3±23.2
Post40	157.0±54.2	75.2±23.2
Pre80	161.3±41.3	87.4±22.7
Post80	164.6±46.9	79.7±22.4
Pre120	162.5±40.2	83.7±29.6
Post120	156.1±38.8	80.6±23.2
Pre-Con	165.8±34.2	80.8±13.7
Post-Con	159.1±42.6	80.5±17.4

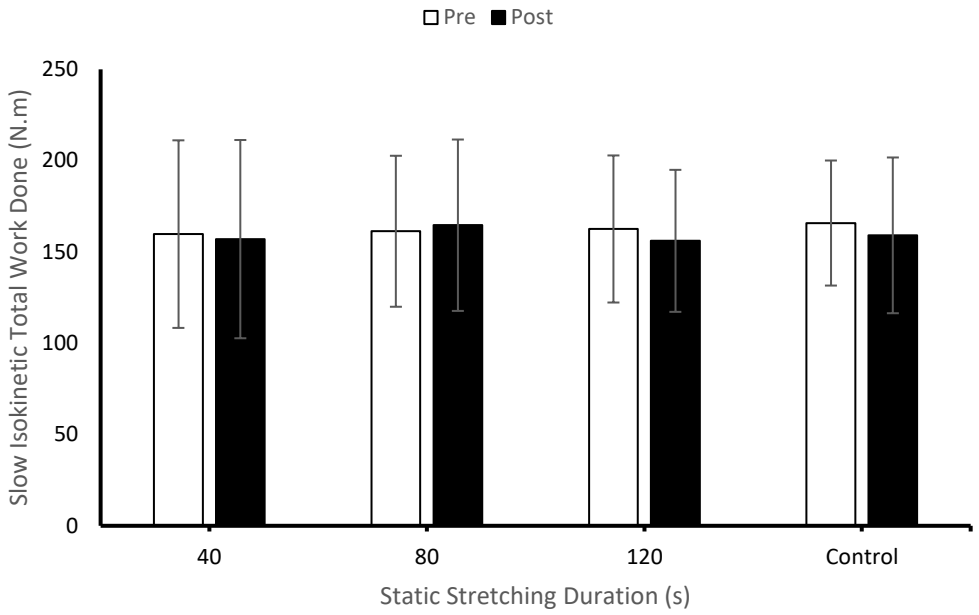

Figure 5: Figure 5 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for fast slow isokinetic total work. There were no significant findings.

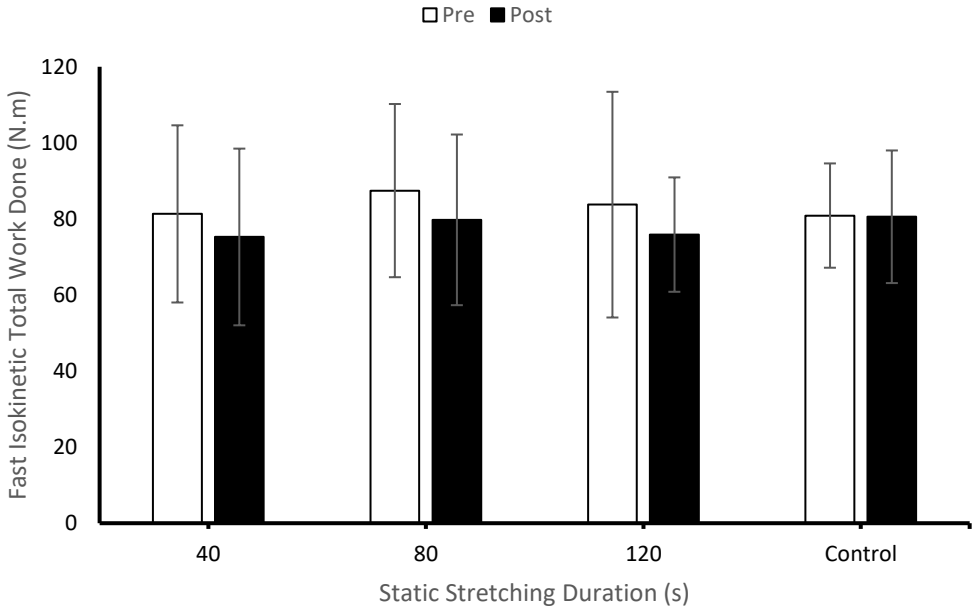

Figure 6: Figure 6 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for fast isokinetic total work. There were no significant findings.

2.2.3 Stiff Leg Drop Jump (SDJ)

The data for SDJ were analyzed for jump height, RSI, and peak power (Table 7). There was no statistically significant interaction of duration*time (Figures 7-9) for any condition with the exception of a main effect for stretch duration for jump height and main effect for time for all the measures. The main effect of stretch duration for jump height was significantly higher in the control session than both 80-s ($p = 0.002$) and 40-s ($p = 0.017$) of antagonist stretching. The main effect of time for all measures decreased significantly from Pre- to Post-test (height: $p = 0.02$, $\eta^2 = 0.31$, RSI: $p < 0.001$, $\eta^2 = 0.36$, peak power: $p = 0.002$, $\eta^2 = 0.49$).

Table 6: Table 6 illustrates Mauchley's test of sphericity and significance values for SDJ height, RSI, and peak power. Shaded boxes highlight significant differences.
Acronyms: Reactive strength index

	Mauchley's Sphericity assumption met		Significance		
	Duration *time	Duration	Duration *time	Duration Main Effect	Time Main Effect
Height	$\chi^2(5) = 7.22$, $p = 0.21$	$\chi^2(5) = 16.63$, $p = 0.005$	$F_{(3, 45)} = 1.02$, $p = 0.39$, $\eta^2 = 0.06$	$F_{(1.8, 27.7)} = 4.95$, $p = 0.01$, $\eta^2 = 0.24$	$F_{(1, 15)} = 6.96$, $p = 0.02$, $\eta^2 = 0.317$
RSI	$\chi^2(5) = 5.43$, $p = 0.36$	$\chi^2(5) = 7.31$, $p = 0.19$	$F_{(3, 45)} = 0.87$, $p = 0.45$, $\eta^2 = 0.05$	$F_{(3, 45)} = 2.59$, $p = 0.06$, $\eta^2 = 0.14$	$F_{(1, 15)} = 19.66$, $p < 0.001$, $\eta^2 = 0.56$
Peak Power	$\chi^2(5) = 9.43$, $p = 0.09$	$\chi^2(5) = 9.28$, $p = 0.09$	$F_{(3, 45)} = 0.21$, $p = 0.89$, $\eta^2 = 0.01$	$F_{(3, 45)} = 2.30$, $p = 0.09$, $\eta^2 = 0.13$	$F_{(1, 15)} = 14.51$, $p = 0.002$, $\eta^2 = 0.49$

Table 7: Mean and standard deviation (mean±SD) data illustrated for all jump height, reactive strength index (RSI), and peak power measures for stiff leg jump height (SDJ).

SDJ	Height	RSI	Peak Power
Pre40	21.2±3.9	0.84±0.22	26.8±5.3
Post40	20.9±4.5	0.81±0.20	25.7±4.7
Pre80	21.4±3.7	0.83±0.21	26.6±5
Post80	21±4.4	0.80±0.21	25.5±5.1
Pre120	21.4±3.6	0.83±0.20	26.4±4.4
Post120	20.9±4.1	0.75±0.21	24.9±5
Pre-Con	22.8±3.9	0.88±0.24	27.7±5.4
Post-Con	21.8±4	0.82±0.28	26.3±6.2

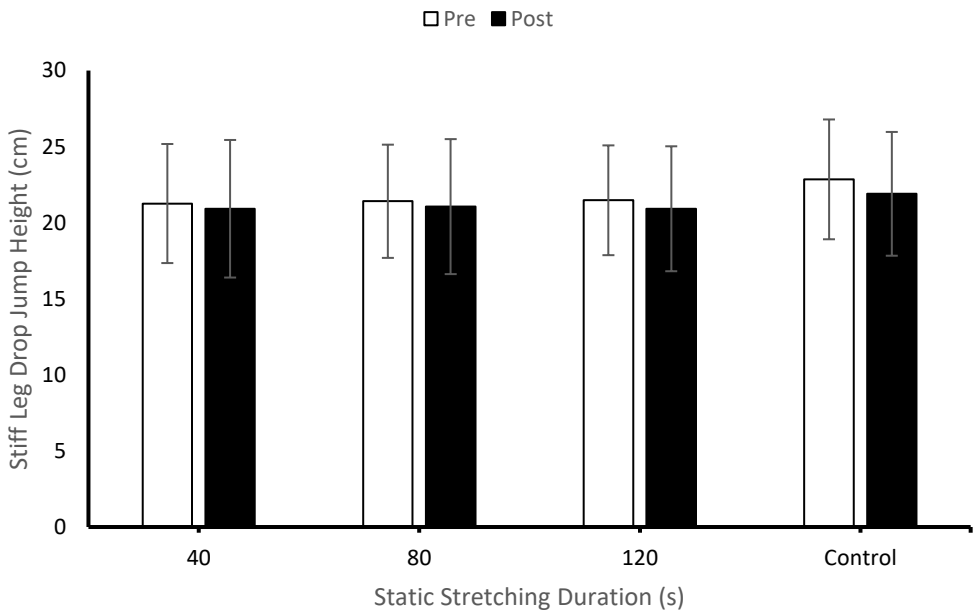

Figure 7: Figure 7 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for stiff leg drop jump height. There were no significant findings.

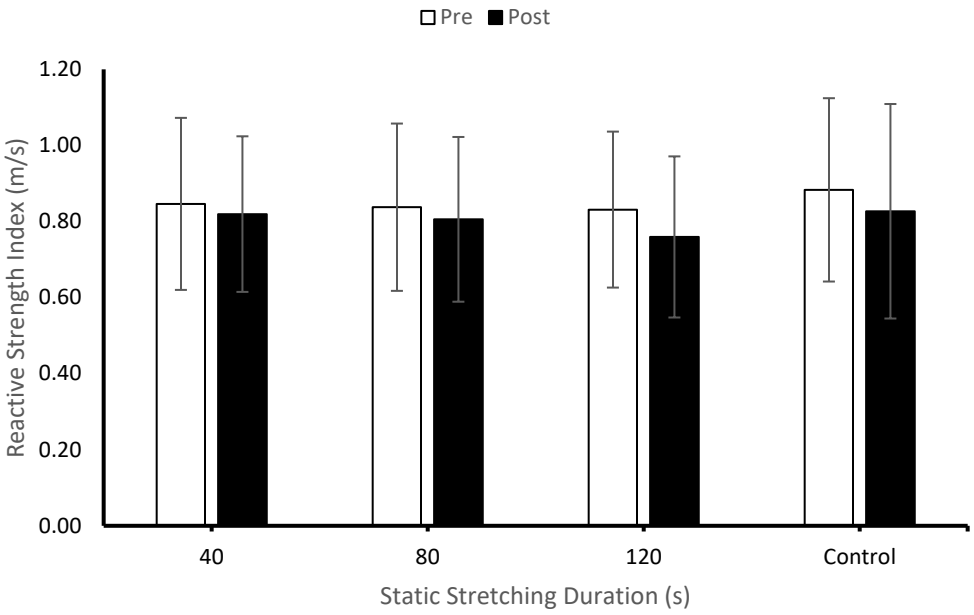

Figure 8: Figure 8 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for reactive strength index. There were no significant findings.

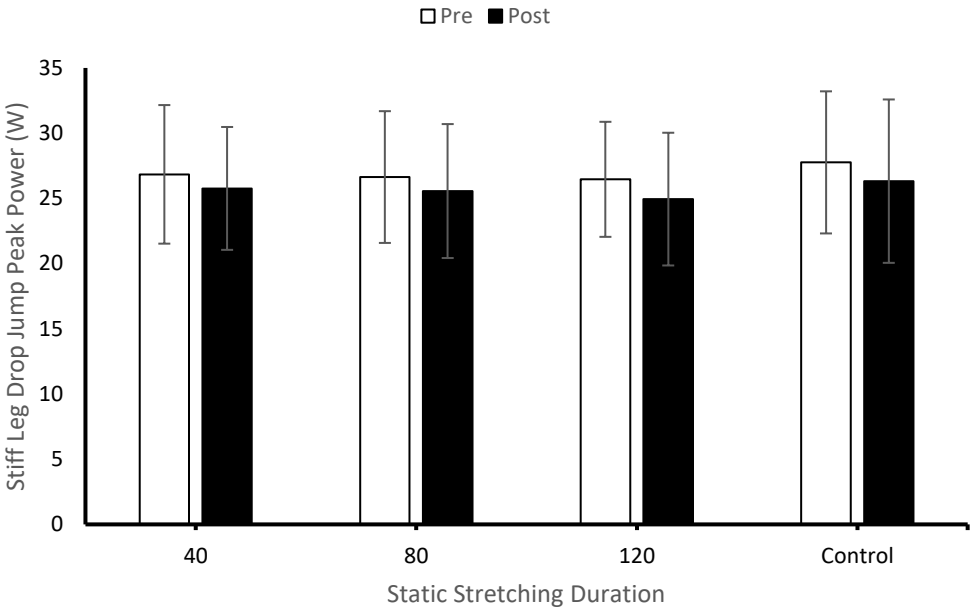

Figure 9: Figure 9 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for peak power. There were no significant findings.

2.2.4 Electromyography

There was no statistically significant interaction of duration*time except for soleus isometric and soleus slow isokinetic EMG where Post120 was significantly lower than Pre120 ($p = 0.002$) and Post-Con was significantly lower than Pre-Con ($p = 0.004$) for soleus isometric and Post120 was significantly lower than Pre120 ($p = 0.002$), Post-Con was significantly lower than Pre-Con ($p = 0.004$) and Post80 was significantly lower than Pre80 ($p = 0.020$) for soleus EMG with slow isokinetic (Figures 10-15).

The main effect of duration was not significant (Table 9) for any condition with the exception of a main effect for the duration for soleus slow isokinetic EMG where the duration of 120 was significantly higher than the duration of 80 ($p = 0.030$). The main effect of time was significant and decreased from pre- to post-test for all the conditions (Table 10).

Table 8: Table 8 illustrates Mauchley's test of sphericity and significance values for EMG. Shaded boxes highlight significant differences.

	Mauchley's Sphericity assumption met		Significance		
	Duration *time	Duration	Duration *time	Duration Main Effect	Time Main Effect
Soleus Isometric	$\chi^2(5) = 4.72$, $p = 0.45$	$\chi^2(5) = 1.08$, $p = 0.95$	$F_{(3, 45)} = 6.12$, $p = 0.001$, $\eta^2 = 0.29$	$(F_{(3, 45)} = 1.657$, $p = 0.19$, $\eta^2 = 0.09$	$F_{(1, 15)} = 13.755$, $p = 0.002$, $\eta^2 = 0.47$
Soleus Slow Isokinetic	$\chi^2(5) = 9.89$, $p = 0.08$	$\chi^2(5) = 1.41$, $p = 0.92$	$F_{(3, 45)} = 4.920$, $p = 0.005$, $\eta^2 = 0.24$	$F_{(3, 45)} = 4.071$, $p = 0.01$, $\eta^2 = 0.21$	$F_{(1, 15)} = 23.878$, $p < 0.001$, $\eta^2 = 0.61$

Soleus Fast Isokinetic	$\chi^2(5) = 8.80$, $p = 0.12$	$\chi^2(5) = 10.947$, $p = 0.053$	$F_{(3, 45)} = 1.857$, $p = 0.15$, $\eta^2 = 0.11$	$F_{(3, 45)} = 0.234$, $p = 0.87$, $\eta^2 = 0.01$	$F_{(1, 15)} = 13.915$, $p = 0.002$, $\eta^2 = 0.48$
Tibialis Anterior Isometric	$\chi^2(5) = 3.49$, $p = 0.62$	$\chi^2(5) = 1.992$, $p = 0.85$	$F_{(3, 45)} = 1.992$, $p = 0.13$, $\eta^2 = 0.11$	$F_{(3, 45)} = 2.539$, $p = 0.06$, $\eta^2 = 0.14$	$F_{(1, 15)} = 5.710$, $p = 0.03$, $\eta^2 = 0.17$
Tibialis Anterior Slow Isokinetic	$\chi^2(5) = 17.09$, $p = 0.004$	$\chi^2(5) = 3.46$, $p = 0.63$	$F_{(1.6, 25.1)} = 2.155$, $p = 0.14$, $\eta^2 = 0.12$	$F_{(3, 45)} = 1.816$, $p = 0.15$, $\eta^2 = 0.11$	$F_{(1, 15)} = 8.454$, $p = 0.01$, $\eta^2 = 0.36$
Tibialis Anterior Fast Isokinetic	$\chi^2(5) = 7.47$, $p = 0.18$	$\chi^2(5) = 4.28$, $p = 0.51$	$F_{(3, 45)} = 1.029$, $p = 0.38$, $\eta^2 = 0.06$	$F_{(3, 45)} = 1.804$, $p = 0.16$, $\eta^2 = 0.10$	$F_{(1, 15)} = 9.435$, $p = 0.008$, $\eta^2 = 0.38$

Table 9: Mean and standard deviation (mean mV±SD) data illustrated for all EMG measures.

	Soleus Isometric	Soleus Slow Isokinetic	Soleus Fast Isokinetic	Tibialis Anterior Isometric	Tibialis Anterior Slow Isokinetic	Tibialis Anterior Fast Isokinetic
Pre40	0.223±0.05	0.222±0.05	0.246±0.06	0.074±0.03	0.084±0.02	0.092±0.03
Post40	0.213±0.04	0.224±0.04	0.227±0.03	0.072±0.02	0.082±0.03	0.088±0.03
Pre80	0.213±0.04	0.224±0.02	0.235±0.03	0.078±0.02	0.094±0.03	0.106±0.03
Post80	0.211±0.03	0.209±0.02	0.236±0.04	0.077±0.02	0.087±0.03	0.095±0.03
Pre120	0.242±0.05	0.260±0.05	0.249±0.04	0.080±0.02	0.100±0.02	0.103±0.03
Post120	0.209±0.04	0.228±0.02	0.235±0.03	0.073±0.02	0.090±0.02	0.095±0.02
Pre-Con	0.221±0.04	0.252±0.04	0.248±0.05	0.069±0.01	0.086±0.02	0.093±0.03
Post-Con	0.202±0.03	0.225±0.03	0.236±0.05	0.061±0.01	0.077±0.02	0.085±0.02

Table 10: Table 8 illustrates the significance and effect sizes for the main effect of time for EMG.

	Significance	Effect Size
Soleus Isometric	0.002	0.478
Soleus Slow Isokinetic	0.001	0.614
Soleus Fast Isokinetic	0.002	0.481
Tibialis Anterior Isometric	0.030	0.276
Tibialis Anterior Slow Isokinetic	0.011	0.360
Tibialis Anterior Fast Isokinetic	0.008	0.386

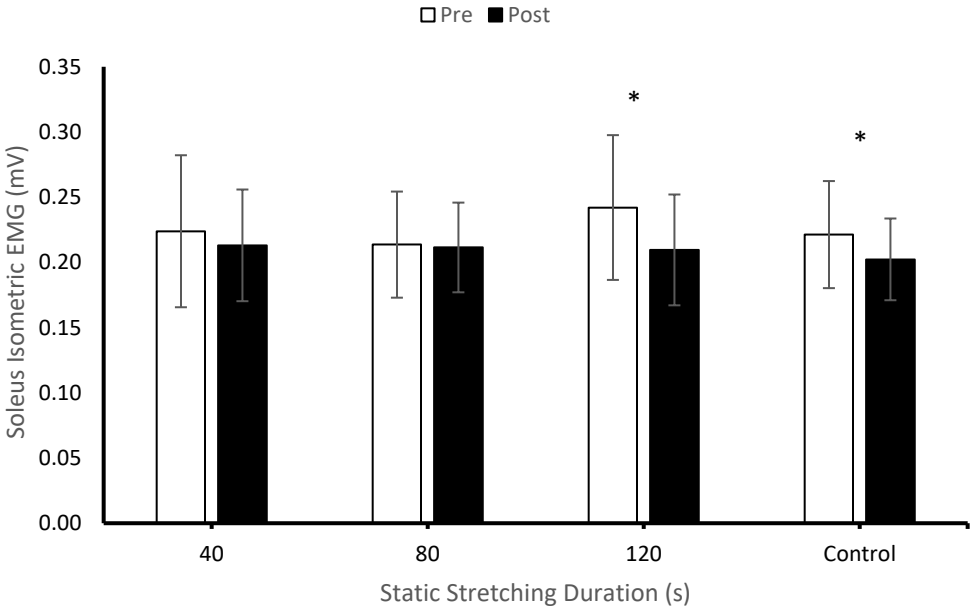

*Figure 10: Figure 10 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for soleus isometric EMG. * indicates significance.*

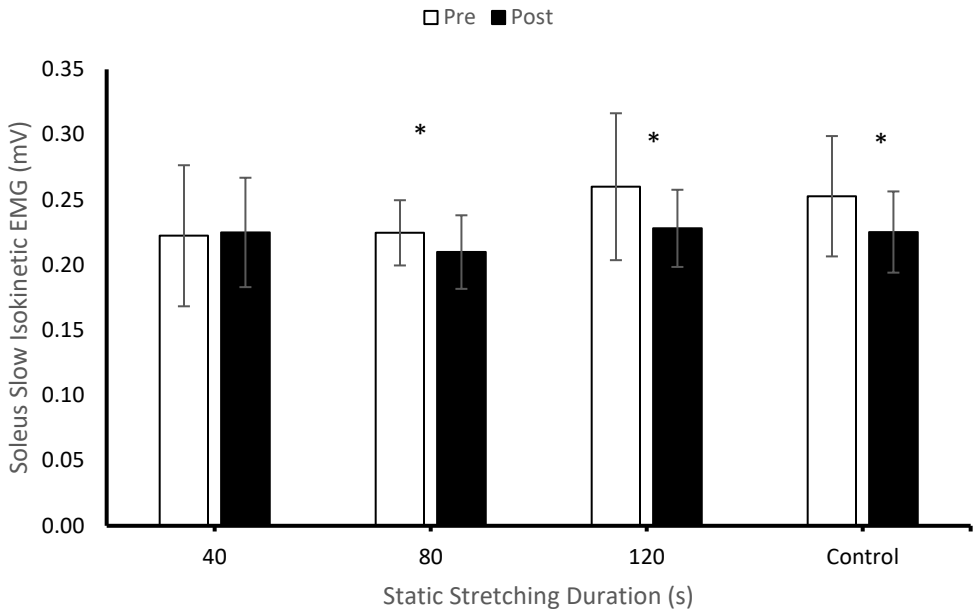

*Figure 11: Figure 11 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for soleus slow isokinetic EMG. * indicates significance.*

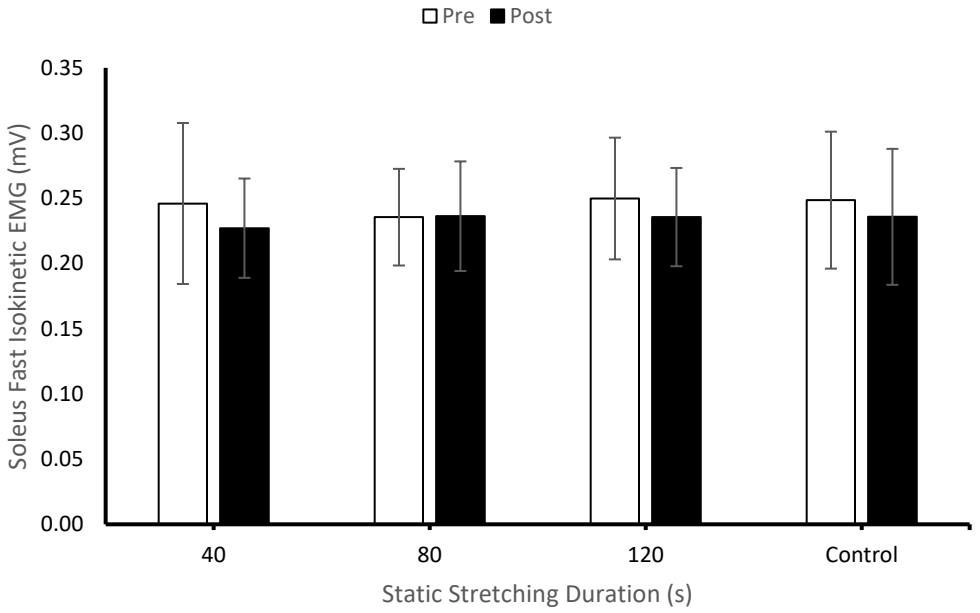

Figure 12: Figure 12 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for soleus fast isokinetic EMG. There were no significant findings.

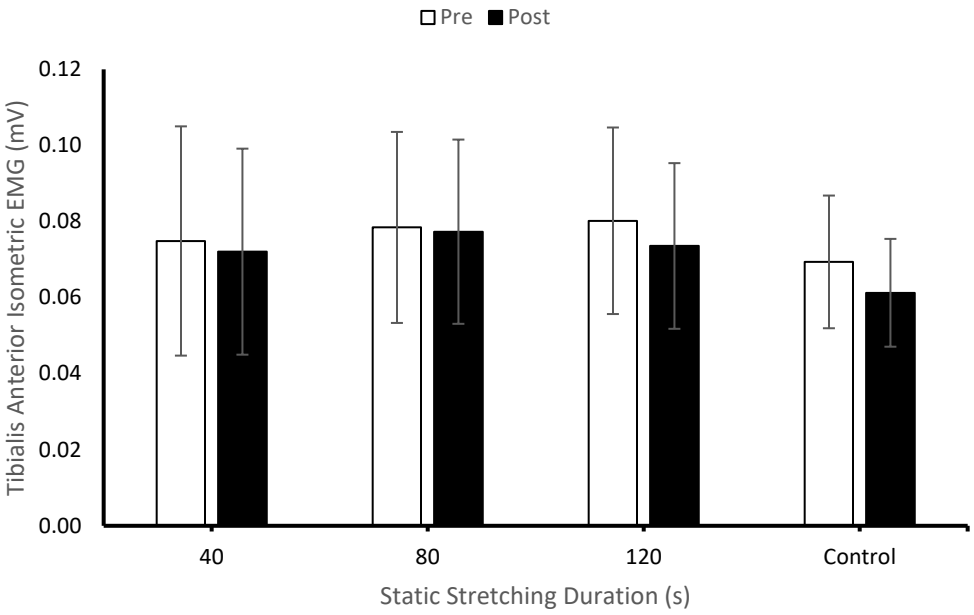

Figure 13: Figure 13 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for Tibialis Anterior Isometric EMG. There were no significant findings.

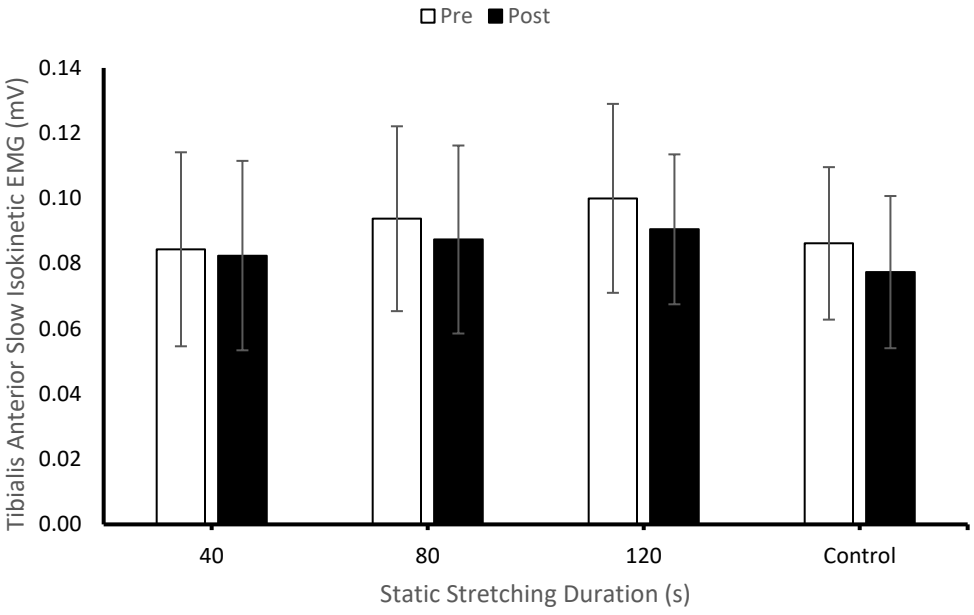

Figure 14: Figure 14 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for Tibialis Anterior slow isokinetic EMG. There were no significant findings.

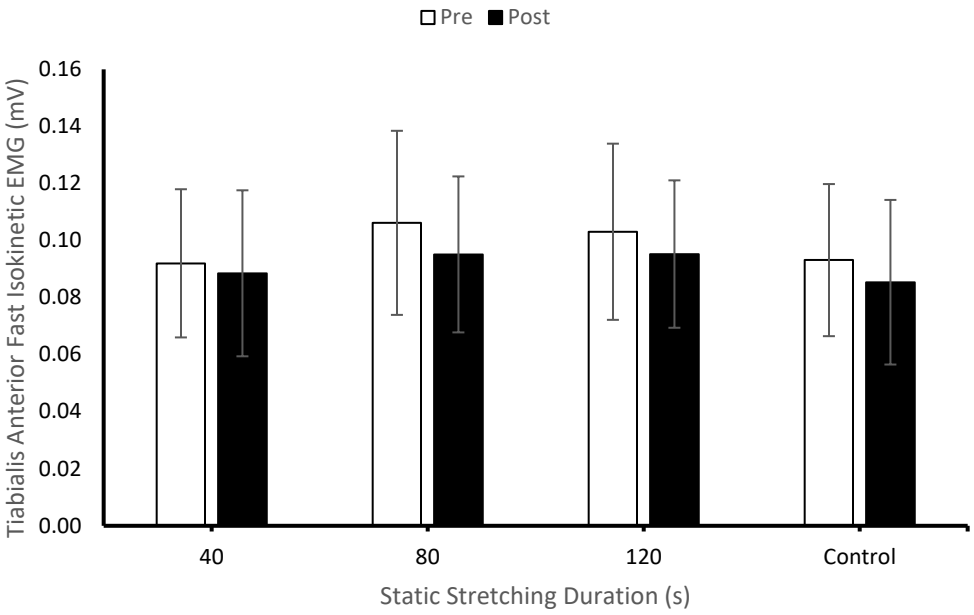

Figure 15: Figure 15 illustrates the interaction of conditions (antagonist stretching for 40-s, 80-s, 120-s and control) and time (pre- and post-test) for Tibialis Anterior fast isokinetic EMG. There were no significant findings.

2.3 Discussion

The major findings of this study were a) there were no effects of specific antagonist stretching durations (40-s, 80-s, 120-s) on pre- to post-intervention isometric peak torque, slow or fast isokinetic peak torque, total work, or SDJ height, RSI and peak power, b) however with all stretch durations and control condition combined (main effect for time) there was an overall decrease in fast and slow isokinetic peak torque, total work as well as all SDJ measures (testing effects), c) SDJ jump heights were lower with 40-s and 80-s of antagonist stretching versus control with no significant change with 120 s and d) soleus EMG decreases were also evident with the control condition suggesting a possibility of testing effects.

In accordance with the present findings, Serefoglu et al. (2017) observed that performing 4 sets of 30s (120s) SS for knee flexors did not significantly affect isokinetic

peak torque or EMG for either slow (60 degrees/s) or fast (240 degrees/s) knee extensions. Similarly, Cogley et al. (2021) conducted a study with 8 sets of 30s of SS (240s) and found no changes in either peak torque or total work, although they did report a significant increase in average power for fast isokinetic (300 degrees/s) contractions. Similar to the 80s of SS in the present study, Sandberg et al. (2012), could not find any significant changes in fast isokinetic peak torque for knee extensors after stretching knee flexors for 90s. To the best of the author's knowledge, this is the first research to investigate the isometric peak torque as well as isokinetic peak torque. Only one study examined the isometric performance, which was TA's MVC and showed no significant difference after stretching plantar flexors for 225s. Jones & Humphrey (2018) explored the impact of stretching hip flexors and dorsiflexors for 90s on VJ height and power and did not find significant differences. VJ did not change significantly after 300s (5x60s) SS of hip flexors, knee flexors, and dorsiflexors either (Montalvo, 2021). Our study also found no significant changes in SDJ performance (height, power, and RSI) across all durations, consistent with the findings of Jones & Humphrey (2018) and Montalvo (2021).

Our current study showed significant reductions in soleus EMG after stretching the TA for 80s and 120s in the slow isokinetic protocol and 120s in the isometric protocol, however, we were not able to show any significant differences in the EMG of either soleus or TA after all other SS durations and testing protocols. Two studies examined the antagonist muscles EMG while doing an isometric protocol and are in agreement with our findings. Sandberg et al. (2012) demonstrated that 90 s of knee flexors SS did not significantly increase knee extensors EMG activity. Similarly, another study by Cè et al. (2021) reported that 240 s of knee flexors SS failed to produce significant changes in the

knee extensors EMG activity. These studies were not able to show any significant changes in the stretched muscle either. Only one study examined the EMG while testing isokinetic peak torque after 240s of SS (8x30s) and did not show any significant changes in either hamstrings after stretching quadriceps or quadriceps after stretching the hamstrings. (Serefoglu et al., 2017). Finally, Sekir et al. (2016) found that 120s of dorsiflexors and hip flexors SS did not affect the plantar flexors and hip extensors EMG activity while doing a VJ test.

Some studies do not support the present EMG findings. For example, both Sandberg et al. (2012) and Wakefield & Cottrell (2015) showed that 90s (3x30s) SS of knee flexors, hip flexors, and dorsiflexors that are all antagonist muscles while doing a VJ resulted in a significant improvement in VJ height and power and also knee extensions fast isokinetic peak torque (300 degrees/s). Also, Cogley et al. (2021) found similar results for knee extensions fast isokinetic peak torque after 240s (8x30s) of stretching the antagonist. In addition, Sekir et al. (2016) showed that 120s of antagonist SS can improve the VJ height substantially although there was no change in EMG results. Three studies applied 40s of pectoralis major SS and found significant improvement in seated row training volume and substantial increase in latissimus dorsi and biceps brachii EMG but no changes in stretched muscles' EMG (Miranda et al., 2015; Paz et al., 2016; Paz et al., 2013). The reasons these studies do not support our results can be attributed to a number of factors. First of all, the antagonist effects of SS might be muscle or muscle size specific. Some studies stretched knee flexors (Sandberg et al., 2012; Wakefield & Cottrell, 2015), while others stretched the pectoralis major (Miranda et al., 2015; Paz et al., 2016; Paz et al., 2013). These muscles are larger (greater volume) than the TA, which was stretched in this study. Another reason

might be the size and strength difference between the agonist and antagonist. TA is not as large and strong as plantar flexors suggesting it may not be able to affect the performance significantly and hence this imbalance might impede antagonist influences compared to larger muscle groups. Furthermore, the stretching method employed in the current study may not have been sufficient to elicit significant changes, as inducing only PF to stretch the TA—which runs diagonally across the tibia from the lateral to medial side diagonally—presents certain challenges. Another factor that might explain the difference is SS duration and intensity. However, based on the studies mentioned above SS ranges from 40s to 240s. Also, only three studies mentioned their SS intensity and all of them explained it as a mild discomfort, however, the precise definition of mild discomfort was not clarified (Sandberg et al., 2012; Wakefield & Cottrell, 2015; Miranda et al., 2015). It should be noted in our current study the SS was performed until the start of discomfort.

The antagonist effects of SS can also be considered as a non-local stretching effect meaning changes in performance and ROM of a muscle or group of muscles that are not stretched (Behm et al., 2021d; Caldwell et al., 2019). The non-local effects of stretching were considered to be of "small magnitude" in the Behm et al. (2021e) review, Cè et al. (2020) found that performing 5 sets of 45 s of unilateral SS of the quadriceps significantly decreased the contralateral knee extensors MVC . In addition, Behm et al. (2021a) revealed that when all performance factors were considered together (including measures of muscle strength, power, and endurance), the overall non-local muscle effect was trivial, suggesting at most only minimal effects. Only when examining the non-local effects in endurance-based studies, there was a moderate effect (Behm et al., 2021a).

Another finding of this study was the SDJ height results were significantly lower during both 40s and 80s sessions compared to the control condition (main effect for stretch durations). The antagonist stretching seem to have more measurable effect on a stretch-shortening activity like the SDJ compared to concentric only action. As these were the main effects for stretch durations the analysis combines both pre- and post-test data. To the author's best knowledge, this is the first study that applied 80s of antagonist SS, however, there are a few studies that examined the effects of 90s (similar duration to 80s) of antagonist SS on VJ height and all found significant increase in VJ height after the SS protocol (Sandberg et al., 2012; Wakefield & Cottrell, 2015). When looking at Shapiro-Wilk test of normality for SDJ height pre- and post80, it gives us $p = 0.280$ and $p = 0.301$ and for pre and post40 are $p = 0.173$ and $p = 0.255$ which means data were normally distributed and there is no outlier finding. Finally, Caldwell et al. (2019) measured single-leg DJ height after unilateral hamstrings stretching for 120s and found that DJ increased significantly for the ipsilateral side.

2.4 Limitations

Whereas, one of the goals was to compare differences between male and female groups there were challenges in recruiting a sufficient number of female participants prevented this objective from being achieved. Another limitation of this study is the large standard deviations relative to the mean values, indicating significant variability among the individual outcomes. Also, there was a limitation while doing SDJ testing. Although we asked all the participants to minimize bending their hip and knee joints to focus more on soleus muscles, it was inevitable to remove the engagement of knee extensors and hip extensors while jumping. An analysis of the gastrocnemius activation may have been

beneficial, however, the soleus will be more predominant in a flexed knee position. Future studies can also look at the neural activity of the gastrocnemius and compare the results with the soleus. Also, since this is the first study that examined the effects of 80s antagonist SS, subsequent studies can investigate how 80s of antagonist SS can affect a different muscle group. Finally, and more importantly, future studies can examine the muscle differences in antagonist stretching effects.

2.5 Conclusions

This study explored the effects of antagonist stretching durations on various performance measures, revealing several key findings. The primary outcome indicated that different durations of antagonist stretching (40s, 80s, 120s) did not significantly influence pre- to post-intervention isometric peak torque, isokinetic peak torque (both slow and fast), total work, or measures of SDJ, including height, RSI, and peak power. However, when all stretch durations and control conditions were combined, there was a general decline in fast and slow isokinetic peak torque, total work, and all SDJ measures, suggesting the presence of testing effects.

A significant reduction in soleus EMG activity was observed during isometric and slow isokinetic protocols following 80s and 120s of stretching, further indicating testing effects. The findings align with previous research that showed no significant changes in peak torque or EMG following various stretching protocols. While some studies reported improvements in performance measures after antagonist stretching, these discrepancies may be due to differences in muscle size, strength, and stretching protocols. Ultimately, two theories can account for the effects of antagonist SS. The first theory is the non-local stretching effect, while the second is antagonist coactivation. When it comes to the

antagonist muscle, these two theories are in opposition to each other. Non-local or crossover effects of stretching happen when stretching a particular muscle or group of muscles leads to alterations in the ROM or performance of another muscle that was not directly stretched although these effects of stretching seem to be small in magnitude (Behm et al., 2021e) which is in agreement with the findings of our current study. On the other side, SS of antagonist muscle, which has been demonstrated to reduce the neural drive to that muscle (Fowles et al., 2000; Robbins et al., 2010), can decrease its force production and coactivation which in turn, can enhance the force generated by the agonist muscle.

2.6 Practical Implications

The findings of this study suggest that incorporating antagonist (dorsiflexors) stretching into pre-exercise routines, especially for durations of 40s, 80s, or 120s, may not significantly enhance PF isometric or isokinetic performance, nor improve jump-related metrics such as SDJ height, RSI, or peak power. In fact, a duration of 80s of antagonist stretching may reduce jump performance. Practitioners should consider these potential effects when designing warm-up protocols, particularly in sports or activities that rely heavily on explosive power and torque. Additionally, the observed decreases in soleus EMG activity indicate that testing effects should be taken into account, suggesting that alternative warm-up strategies or different stretching protocols may be more beneficial for optimizing performance. Coaches and athletes may need to reassess the role of antagonist stretching in their routines, potentially favoring other methods that better support performance goals.

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Appendix:

Appendix 1- Ethics Approval Letter

MEMORIAL UNIVERSITY

Interdisciplinary Committee on
Ethics in Human Research (ICEHR)

St. John's, NL Canada A1C 5S7
Tel: 709 864-2561 icehr@mun.ca
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ICEHR Number:	**20241815-HK**
Approval Period:	April 9, 2024 – April 30, 2025
Funding Source:	NSERC [RIS# 20231244]
Responsible Faculty:	Dr. David Behm School of Human Kinetics and Recreation
Title of Project:	*Effects of Antagonist Stretching on Agonist Performance*
Amendment #:	***01***

April 24, 2024

Mr. Mohammadmahdi Bahrami
School of Human Kinetics and Recreation
Memorial University

Dear Mr. Bahrami:

The Interdisciplinary Committee on Ethics in Human Research (ICEHR) has reviewed the proposed revisions for the above referenced project, as outlined in your amendment request dated April 17, 2024. We are pleased to give approval to the revised protocols, as described in your request, provided all other previously approved protocols are followed.

The *TCPS2* **requires** that you **strictly adhere to the protocol and documents as last reviewed** by ICEHR. If you need to make any other additions and/or modifications during the conduct of the research, you must submit an Amendment Request with a description of these changes, for the Committee's review of potential ethical issues, before they may be implemented. Submit a Personnel Change Form to add or remove project team members and/or research staff. Also, to inform ICEHR of any unanticipated occurrences, an Adverse Event Report must be submitted with an indication of how the unexpected event may affect the continuation of the project.

Your ethics clearance for this project expires **April 30, 2025**, before which time you must submit an Annual Update to ICEHR, as required by the *TCPS2*. If you plan to continue the project, you need to request renewal of your ethics clearance, and include a brief summary on the progress of your research. When the project no longer requires contact with human participants, is completed and/or terminated, you need to provide an annual update with a brief final summary, and your file will be closed.

All post-approval ICEHR event forms noted above must be submitted by selecting the *Applications: Post-Review* link on your Researcher Portal homepage.

The Committee would like to thank you for the update on your proposal and we wish you well with your research.

Yours sincerely,

Alyson Byrne

Alyson Byrne, Ph.D.
Vice-Chair, Interdisciplinary Committee on
Ethics in Human Research

AB/bc

cc: Supervisor – Dr. David Behm, School of Human Kinetics and Recreation