Roller massage decreases spinal excitability to the soleus

by

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ABSTRACT

Evidence suggests that foam rolling and roller massage can increase range of motion and alter pain perception. These effects have been shown not only in the target muscle, but also in the contralateral homologue that was not involved in the intervention. The global nature of these effects suggests that the benefits of rolling are primarily accrued through neural responses and not connective tissue. Therefore, the primary objective of this thesis was to investigate the underlying mechanisms responsible for improvements in range of motion and altered pain perception during and after roller massage. This required the use of a sensitive neurophysiological measure that is subject to modulation via afferent input. The results of the present study serve as the first evidence of neural modulation of spinal excitability during roller massage. It is plausible that immediate changes in range of motion and pain perception following brief roller massage can be attributed to the observed inhibition at the spinal level.

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LIST OF ABBREVIATIONS

ANOVA	Analysis of Variance
cm	Centimeter
dB	Decibel
EMG	Electromyography
FR	Foam Rolling
GTO	Golgi Tendon Organ
Hz	Hertz
H_{max}	Maximum H-Reflex
H-Reflex	Hoffmann Reflex
ICC	Interclass Correlation Coefficient
kg	Kilogram
mA	Milliamp
M_{max}	Maximum M-Wave
ms	Millisecond
mV	Millivolt
PF	Plantar Flexors
P-P	Peak to Peak
PPT	Pain Pressure Threshold
RM	Roller Massage
ROM	Range of Motion
SD	Standard Deviation
SEM	Standard Error of Measurement
SMR	Self Myofascial Release
TA	Tibialis anterior
VAS	Visual Analog Scale

Chapter 1: Review of Literature

1.1 Introduction

Foam rolling (FR) and roller massage (RM) are extensively used in exercise and sport practice. Prior research suggests these rolling devices may be effective interventions for increasing joint range of motion (ROM) and improving pre- and post-exercise performance. This popular practice stems from the direct comparison to a manual therapy technique known as myofascial release. Myofascial release involves applying low load, long duration mechanical force to manipulate the myofascia and is intended to restore optimal length, decrease pain, and improve function (Barnes, 1990). Often called self-myofascial release (SMR), the simplistic design and practical application of FR and RM can act as an alternative or adjunct to massage therapy. The most common tools used for SMR are the foam roller (Cavanaugh, Aboodarda, Hodgson, & Behm, 2016; Cheatham & Kolber, 2017; Cheatham, Kolber, & Cain, 2017; Cheatham, Kolber, Cain, & Lee, 2015; Kelly & Beardsley, 2016; Macdonald, Button, Drinkwater, & Behm, 2014; Macdonald et al., 2013; Monteiro & Neto, 2016; Pearcey et al., 2015; Vaughan & McLaughlin, 2014), roller massager (Aboodarda, Spence, & Button, 2015; Bradbury-Squires et al., 2015; Casanova et al., 2017; Cavanaugh, Doweling, et al., 2016; Grabow et al., 2017; Halperin, Aboodarda, Button, Andersen, & Behm, 2014; Jay et al., 2014; Mikesky, Bahamonde, Stanton, Alvey, & Fitton, 2002; Monteiro, Cavanaugh, Frost, & Novaes, 2016; K. M. Sullivan, Silvey, Button, & Behm, 2013) and tennis ball (Grieve et al., 2015). While these rolling devices provide increased portability and accessibility compared to massage therapy, there is controversy in the literature as to whether they actually release myofascia or whether the improvements are primarily accrued through neural responses.

1.2 Fascia

Fascia is defined as fibrous collagenous tissues, which are part of a body wide tensional force transmission system (Schleip, Jager, & Klingler, 2012). The current models of fascial plasticity include properties of thixotropy and piezoelectricity. Thixotropy was originally defined as a transformation in density following the application of heat or mechanical pressure (Juhan, 1987). Piezoelectricity refers to an increased rate of collagen synthesis as a result of altered electrical properties following mechanical deformation of fascial tissue (Juhan, 1987). While these mechanical models can account for long-term changes in plasticity, they do not explain more immediate changes in tissue quality (Schleip, 2003). One plausible explanation for the immediate tissue changes is that they are primarily neural adaptations. There is evidence to suggest a number of intrafascial mechanoreceptors are intimately linked with the autonomic nervous system (Coote & Perez-Gonzalez, 1970; Johansson, Sjölander, & Sojka, 1991; Mitchell & Schmidt, 1983). Schleip (2000) proposed that feedback from mechanoreceptors enables the nervous system to function as a complex, self-regulatory field with continual reorganization. These findings indicate that neurophysiological influences, in addition to thixotropy and piezoelectricity, merit inclusion in an explanatory model of fascial plasticity.

1.2.1 Fascial Receptors

Fascia is densely populated with three groups of mechanoreceptors: type Ib Golgi tendon organs (GTO), type II Pacini corpuscles and Ruffini endings, and type III/IV interstitial myofascial tissue receptors. While GTO are known to respond to muscular contraction and rapid changes in muscle length (Jami, 1992), Pacini corpuscles and Ruffini organs are sensory receptors that respond to rapid changes in pressure, and vibration (Schleip, 2003). Ruffini organs also appear to be responsive to sustained pressure and are believed to inhibit sympathetic nervous system activity (Van den Burg & Capri, 1999). The most abundant of all intrafascial mechanoreceptors, type III and IV sensory nerves are comprised of primarily unmyelinated free nerve endings responsive to changes in pain and mechanical tension or pressure (Schleip, 2003). It is likely that myofascial release activates a variety of these intrafascial mechanoreceptors, though the exact mechanisms through which this occurs remain unclear.

In addition to fascial mechanoreceptors, myofascial release also stimulates cutaneous mechanoreceptors. Johnson (2001) highlights the apparent specialization of each of the four primary cutaneous mechanoreceptive afferent neuron types. In the human hand, Merkel cells and what are likely Ruffini organs are considered groups of slowly adapting receptors. Merkel cells are responsible for spatial resolution of a tactile stimulus and provide information regarding the shape of the stimulus, while the second group of slowly adapting receptors has been shown to respond to skin stretch (Johnson, 2001). It has been suggested that Merkel cells work in conjunction with either proprioceptive organs (GTO) or groups of rapidly adapting receptors to transmit afferent input in response to tactile stimuli (Johnson, 2001). Pacini corpuscles and Meissner corpuscles comprise the final two groups of primary cutaneous mechanoreceptors. These groups are rapidly adapting, highly sensitive, and relatively large structures with many layers protecting their extreme sensitivity from mechanical pressure and strain (Johnson, 2001). All sensory afferent neurons described above have been shown to have central projections to either the brain or spinal cord and may be involved in mediating muscle tone and joint stiffness (McGlone & Reilly, 2010). Therefore, it is likely that stimulation of intrafascial and cutaneous mechanoreceptors will lead to altered proprioceptive and afferent input to either the brain or spinal cord, which may explain palpable changes in tissue quality following myofascial release.

1.3 Massage Therapy

Cafarelli and Flint (1992) defined massage as mechanical manipulation of body tissues with rhythmical pressure and stroking for the purpose of promoting health and well-being. A number of massage therapy techniques are common in rehabilitation and sport practice, including effleurage, petrissage, tapotement, and myofascial release (Weerapong, Hume, & Kolt, 2005). Massage is believed to benefit athletes through its biomechanical, physiological, neurological, and psychological mechanisms (Weerapong et al., 2005).

1.3.1 Massage and Performance

Massage therapy is used both pre- and post-event in an effort to improve athletic performance (Callaghan, 1993). Despite widespread use in elite level athletics, evidence for the physiological and psychological benefits of massage therapy interventions is weak. Weerapong et al. (2005) reported that the majority of studies have examined either the psychological effects of massage or the effect of post-exercise massage on muscle soreness, with inconclusive findings for both. Both Brummitt (2008) and Weerapong et al. (2005) indicate a lack of well-controlled studies as the limiting factor in the interpretation of findings in the current literature. In terms of pre-exercise massage, Goodwin et al. (2007) found that a combination of massage techniques including effleurage, petrissage, and tapotement did not negatively affect subsequent sprint performance. Arabaci (2008) found that a similar protocol resulted in increased ROM immediately following the massage; however, these improvements were coupled with detriments in reaction time, vertical jump, and sprint performance. While Goodwin et al. (2007) did not assess ROM, they did include a full dynamic warm-up immediately following the massage therapy intervention. It is likely that any massage-induced decrements in performance would have been mitigated by the dynamic warm-up, similar to findings in the static stretching literature (Behm, Blazevich, Kay, & McHugh, 2015). Further research is required to evaluate the effectiveness of massage therapy interventions for improving athletic performance, muscle recovery, and injury prevention.

1.3.2 Massage and Pain

Massage is believed to alter pain perception and is commonly used in the treatment of both chronic and acute pain. While the exact mechanism of pain reduction is unknown, there are several dominant theories in the literature. One theory is that massage-like mechanical pressure provides analgesic effects via the ascending or descending pain inhibitory systems, or through stimulation of the autonomic nervous system (Melzack & Wall, 1965; Moayedi & Davis, 2013; Weerapong et al., 2005). Activation of mechanoreceptors and nociceptors can alter the transmission of ascending nociceptive information and result in a localized analgesic response (Moayedi & Davis, 2013). Alternatively, it is possible that ascending nociceptive information can result in monoaminergic inhibition of pain, which will result in a diffuse or global response leading to a reduction in pain perception throughout the system (Sigurdsson & Maixner, 1994). Mense (2000) suggested that the descending anti-nociceptive system has a great influence on input from nociceptors located in both muscle and skin. It is also believed that massage therapy can stimulate an increase in parasympathetic activity, characterized by changes in biochemical substances such as serotonin, cortisol, endorphin and oxytocin (Weerapong et al., 2005). Aboodarda et al. (2015) suggested that parasympathetic reflexes may contribute to the release of stress from myofascial tissue by relaxing, releasing, or inhibiting the strain on the smooth muscle and subsequently decreasing levels of perceived pain. No matter the theory, pain is highly subjective and the interaction between nociceptive input and pain perception is complex (Wiech, Ploner, & Tracey, 2008). Thus, it is likely that a combination of ascending and descending afferent input from the brain, spinal cord, and periphery are involved in the modulation of pain perception following massage.

1.4 Foam Rolling and Roller Massage

While it is believed that FR and RM may benefit users through similar mechanisms as massage therapy or myofascial release, to date, there are no studies that directly compare these interventions. Prior research demonstrates that FR and RM can increase range of motion (ROM) (Bradbury-Squires et al., 2015; Grabow et al., 2017; Kelly & Beardsley, 2016; Macdonald et al., 2014; Macdonald et al., 2013; Monteiro et al., 2016; Monteiro & Neto, 2016; Monteiro et al., 2017; Sullivan et al., 2013), as well as either increase (Macdonald et al., 2014; Peacock, Krein, Silver, Sanders, & Von Carlowitz, 2014) or maintain subsequent force output (Casanova et al., 2017; Grabow et al., 2017; Macdonald et al., 2013; Pearcey et al., 2015; Sullivan et al., 2013), and improve neuromuscular efficiency (Bradbury-Squires et al., 2015). In addition to its effects on performance measures, evidence suggests that FR and RM can increase blood flow (Hotfiel et al., 2016; Okamoto, Masuhara, & Ikuta, 2014), diminish evoked pain (Cavanaugh, Doweling, et al., 2016), and reduce pain associated with muscle tender points (Aboodarda et al., 2015) and exercise-induced muscle damage (Casanova et al., 2017; Jay et al., 2014; Macdonald et al., 2014; Pearcey et al., 2015; Vaughan & McLaughlin, 2014). While the outcomes of FR and RM have been, and continue to be examined, there is currently no accepted mechanism to explain how adaptations are accrued.

1.4.1 Mechanisms - Range of Motion

Although there are no studies that have directly examined the mechanisms responsible for the reported increases in ROM following FR or RM, there are several theories. A common hypothesis is that changes in both tissue compliance and the musculotendinous unit may be responsible (Halperin et al., 2014; Macdonald et al., 2013; Sullivan et al., 2013). It has also been suggested that the benefits of RM may be accrued through neural responses in addition to changes in connective tissue (Macdonald et al., 2014). Kelly & Beardsley (2016) demonstrated a FR-induced increase in ROM of both the ipsilateral and contralateral plantar flexors (PF); the effects remained for at least 10-min post-intervention. It was suggested that an increase in rolling tolerance, similar to the theory of stretch tolerance (Magnusson et al., 1996), may be responsible for these improvements. Furthermore, Chaudhry et al. (2008) demonstrated that forces beyond normal physiological ranges are required to produce compression and shear in the plantar fascia. These findings support the notion that the benefits of rolling may be accrued through neural responses rather than changes tissue compliance and the musculotendinous unit. It is unlikely that a change in viscoelastic properties or mechanical deformation of tissues is possible during short duration or moderate intensity rolling (Schleip, 2003). Thus, it is probable that reported increases in ROM may not be due entirely to local muscular changes. Perhaps neural mechanisms, such as a change in spinal excitability due to increased afferent input, are responsible for mediating improvements in ROM following FR and RM interventions, although this has yet to be demonstrated in the literature.

1.4.2 Mechanisms - Pain

It is well documented that FR and RM interventions can alter the perception of pain (Aboodarda et al., 2015; Bradbury-Squires et al., 2015; Casanova et al., 2017; Cavanaugh, Doweling, et al., 2016; Cheatham & Kolber, 2017; Jay et al., 2014; Macdonald et al., 2014; Pearcey et al., 2015; Vaughan & McLaughlin, 2014). Recent evidence demonstrates an increase in pain pressure threshold (PPT) to both the ipsilateral and contralateral limbs (Aboodarda et al., 2015; Cavanaugh, Doweling, et al., 2016; Cheatham & Kolber, 2017; Cheatham et al., 2017).

Whereas it is possible that changes in PPT of the ipsilateral limb can be attributed to stimulation of fascial mechanoreceptors, changes in PPT observed in the contralateral limb indicate the involvement of a global neural mechanism. Similar to massage therapy, it is believed that a central pain modulatory system plays the main role in mediating perceived pain following FR and RM (Aboodarda et al., 2015).

It is interesting to note that pain perception is often used to quantify the intensity of pressure exerted on the participant in FR and RM studies. Therefore, these studies inherently include a noxious stimulus and often use a sham or light touch condition in addition to a true experimental control condition. Recent evidence highlights the importance of such an experimental group in FR and RM studies. Aboodarda et al. (2015) observed a decrease in PPT of the plantar flexors in the sham (light RM) condition while heavy RM resulted in an increase in PPT of both the ipsilateral and contralateral limbs. Since light RM was not a noxious stimulus, the authors hypothesized that the decreased PPT following this intervention may be associated with increased sensitivity of superficial nociceptors. Similarly, Cavanaugh et al. (2016) reported an increase in pain perception in the sham condition following evoked pain that was not revealed during the heavy RM condition. That is, the presence of a noxious stimulus attenuated the increase in pain perception observed in both sham and control conditions. These findings suggest that a sham condition should be included in any further investigations of neurophysiological responses to FR and RM.

Reported increases in ROM and PPT following FR and RM interventions cannot be conclusively attributed to local muscular changes. Several authors have suggested the possibility of altered neuromuscular excitability during and/or following rolling interventions (Bradbury-Squires et al., 2015; Macdonald et al., 2013; Sullivan et al., 2013); however, it remains unclear whether a neurophysiological mechanism, such as neural inhibition at the spinal level due to changes in afferent input, is responsible for mediating FR- and RM-induced increases in ROM and PPT. Evaluating neural responses to FR and RM will require the use of a neurophysiological measure that is subject to modulation via afferent input.

1.5 The H-Reflex

First described by Paul Hoffmann (1910; 1918) the Hoffmann (H-) reflex is of particular interest in the assessment of spinal excitability and is considered one of the most extensively studied neurophysiological measures (for review see Misiaszek, 2003; Zehr, 2002). Characterized by primarily monosynaptic projections of the group Ia afferents onto the homonymous motoneurones, the neural circuitry responsible for the H-reflex is similar to the monosynaptic stretch reflex (Misiaszek, 2003). As such, it is often described as the electrical analogue to the stretch reflex; however, the H-reflex bypasses the effects of both the muscle spindle and gamma motoneurones. Described as one of the most confounding assumptions in human reflex studies (Knikou, 2008), many researchers continue to operate under the assumption that the H-reflex is a purely monosynaptic reflex. The duration of the H-reflex suggests the potential for oligosynaptic contributions to the latter portion of the H-reflex must be considered (Knikou, 2008; Zehr, 2002). Additionally, the influence of presynaptic inhibition, homosynaptic depression, and the intrinsic excitatory properties of the alpha motoneurones must also be considered before conclusions may be drawn regarding changes in excitability at the spinal level (Knikou, 2008; Palmieri, Ingersoll, & Hoffman, 2004; Zehr, 2002).

Evoked by low intensity stimulation of a peripheral nerve, the relative ease with which the H-reflex can be elicited from numerous muscle groups in both the upper and lower limbs makes it an attractive tool for clinical and research purposes (Misiaszek, 2003; Zehr, 2002).

Stimulation of the peripheral nerve will evoke action potentials in both afferent and efferent nerves. When stimulation delivered to the nerve is above threshold for activation of Ia afferents, action potentials propagate toward the spinal cord. Sufficient Ia afferent activity will depolarize the afferent terminals and release neurotransmitters into the synaptic cleft at the Ia-alpha motoneurone synapse (Zehr, 2002). This results in post-synaptic depolarization of the alpha motoneurones, generating action potentials that cause neurotransmitter release at the neuromuscular junction. This neurotransmitter causes depolarization and synchronized twitch of the muscle fibres, which will be recorded as an H-reflex in the muscle under study (Palmieri et al., 2004; Zehr, 2002).

Activation of efferent nerves results in a direct motor response, known as the muscle action potential (M-) wave. Action potentials travel orthodromically toward the muscle and can be recorded as the M-wave before they travel antidromically toward the spinal cord and the Ia-alpha motoneurone synapse (Palmieri et al., 2004; Zehr, 2002). As the stimulation intensity increases, the magnitude of the direct efferent motor response will increase. This results in a collision of afferent and efferent information known as an antidromic collision (Magladery & McDougal, 1950). The increasing magnitude of the antidromic efferent response will collide with the orthodromic afferent response and effectively reduce the magnitude of the H-reflex recorded from the muscle of interest (Palmieri et al., 2004; Zehr, 2002). Eventually, the H-Reflex will be abolished and the motor response will reach a maximum value, known as M-max. This value represents the maximal activation of the motoneurone pool and allows for normalization of H-reflex responses as a percentage of motoneurone pool activation (Pierrot-Deseilligny & Mazevet, 2000).

1.5.1 Reflex Modulation

The H-reflex is subject to modulation by a number of confounding factors, including the influence of presynaptic inhibition and homosynaptic depression (McNeil, Butler, Taylor, & Gandevia, 2013; Misiaszek, 2003). Though often reported as a measure of motoneurone excitability, it is more appropriately used as a measure of changes in presynaptic inhibition at the Ia-alpha motoneurone synapse. If all confounding factors have been minimized through sound experimental protocols, the H-reflex serves as a valuable tool to measure the excitability of the neural components of the reflex arc at the spinal level (Zehr, 2002).

Presynaptic Inhibition

The degree of presynaptic inhibition of Ia terminals has long been known to alter the size of the H-reflex (for review see Rudomin & Schmidt, 1999). It is imperative that levels and sources of extraneous afferent input must be monitored during investigations involving the H-reflex in order to minimize presynaptic inhibitory responses (Palmieri et al., 2004). Originally described in animal work from 1957, (Frank & Fuortes) reported a depression of monosynaptic excitatory potentials that occurred in the absence of change in motoneurone excitability or postsynaptic potential. Rudomin and Schmidt (1999) stated that the presynaptic inhibitory synapses of afferent terminals are the primary targets for all nervous activity aimed at inhibiting afferent input in vertebrates. Therefore, activation of remote muscle afferents via application of FR or RM is likely to cause presynaptic inhibition of the Ia afferent and presynaptic suppression of the H-reflex (Misiaszek, 2003).

Homosynaptic Depression

The history of activity at the Ia-alpha motoneurone synapse is an important factor affecting the transmission of subsequent afferent input. Any prior activation of the Ia afferent will lead to a reduction of available neurotransmitter stores at the Ia terminal (Misiaszek, 2003). It is preferred that stimulations of the peripheral nerve be delivered at a frequency of 0.1 Hz to avoid the influence of homosynaptic depression at the Ia terminal (McNeil et al., 2013). However, as the decay of homosynaptic depression is curvilinear (Magladery & McDougal, 1950), stimulating at 0.2–0.3 Hz strikes an acceptable balance between the level of depression and the time required to collect the responses (Pierrot-Deseilligny & Mazevet, 2000).

FR and RM at their most basic form are nothing more than doses of afferent input delivered to the nervous system at varying amplitudes, frequencies, and intensities. The H-reflex is subject to modulation via afferent input and is a highly sensitive neurophysiological measure that can provide valuable insight into spinal-mediated changes in the excitability of the neuromuscular system (Behm et al., 2013). It is plausible that an investigation of spinal excitability during FR and RM will elucidate a mechanism responsible for the numerous reports of changes in range of motion and pain tolerance following rolling interventions.

1.6 Massage and the H-Reflex

To investigate the effects of FR and RM techniques on spinal reflex excitability, the closest parallel from which to draw is a massage-induced change in H-reflex response. The bulk of the literature is focused on the soleus, due to the large separation of stimulus thresholds of its H-reflex and M-wave (Capaday, 1997) and the accessibility of the superficial posterior tibial nerve. Several authors have reported decreases in soleus H-reflex amplitude from a number of massage techniques and intensities (Belanger, Morin, Pepin, Tremblay, & Vachon, 1989; Goldberg, Seaborne, Sullivan, & Leduc, 1994; Goldberg, Sullivan, & Seaborne, 1992; Morelli, Chapman, & Sullivan, 1999; Morelli, Seaborne, & Sullivan, 1990; Morelli, Sullivan, & Chapman, 1998; Sullivan, Williams, Seaborne, & Morelli, 1991). The techniques that have shown a significant decrease in H-reflex amplitude include: petrissage, tapotement and musculotendinous junction massage. It appears as though the changes are pressure-dependent, with a deeper massage having a greater inhibitory effect on spinal excitability (Behm et al., 2013; Goldberg et al., 1992; Sullivan et al., 1991). Tapotement massage, a more vigorous technique, produced greater reductions in H-reflex response when compared to musculotendinous junction massage (Behm et al., 2013). These authors propose a vibration-like effect from the tapotement technique, which has been shown to reduce spinal excitability (De Gail, Lance, & Neilson, 1966; Martin, Roll, & Gauthier, 1986; Sayenko, Masani, Alizadeh-Meghrazi, Popovic, & Craven, 2010) due to presynaptic inhibition of Ia afferents (Lapole, Deroussen, Pérot, & Petitjean, 2012).

The location and duration of the massage intervention has been previously analyzed and despite conflicting evidence on location, it has been shown that duration plays a minimal role in inhibiting spinal excitability. For example, soleus H-reflex amplitudes remained unchanged during petrissage of the ipsilateral and contralateral hamstrings, as well as the contralateral plantar flexors. This indicates a muscle-specific response and suggests neural mediation of spinal excitability may be specific to the homonymous motoneurone pool (Sullivan et al., 1991). Conversely, Morelli et al. (1998) evaluated the medial gastrocnemius H-reflex response during petrissage of the ipsilateral soleus. A marked reduction in spinal excitability to the medial gastrocnemius was reported, indicating that the effects may not be limited to the homonymous motoneurone pool, but rather extend to synergist muscles in close proximity (Morelli et al., 1998). A similar reduction in soleus H-reflex amplitude has been previously observed in a study examining muscle tapping of various sites on the ipsilateral limb (Belanger et al., 1989); however, it has been suggested that muscle tapping activates similar mechanisms to tapotement, which has shown a greater inhibitory effect than petrissage (Behm et al., 2013). Given the conflicting evidence, it is difficult to speculate as to the degree of specificity associated with spinal reflex inhibition.

It does not appear as though the duration of the massage intervention is particularly important. Decreases in spinal excitability observed in as little as 30-seconds (Behm et al., 2013), as long as 4-minutes (Sullivan et al., 1991), and most commonly approximately 3-minutes (Goldberg et al., 1992; Morelli et al., 1999; Morelli et al., 1990; Morelli et al., 1998). Regardless of the duration used, no prolonged decreases in spinal excitability were reported. Morelli and colleagues (Goldberg et al., 1994; Goldberg et al., 1992; Morelli et al., 1999; Morelli et al., 1990; Morelli et al., 1998; Sullivan et al., 1991) have provided the bulk of the literature regarding changes in spinal excitability during and after massage therapy. These authors postulate that stimulation of mechanoreceptors leads to altered afferent input and a marked reduction in spinal excitability to the affected muscle. In support, Schleip (2003) suggests that fascia is densely innervated by mechanoreceptors and therefore the manual stimulation or myofascial release may lead to changes in muscle tone. The fascial mechanoreceptors are likely influenced by both gamma and alpha motoneurones, which affect tissue viscosity (Schleip, 2003). This may be one explanation for the observed increases in ROM following massage interventions and is likely to extend to RM and FR modalities.

Deep massage has shown a more pronounced inhibitory effect suggesting that deep mechanoreceptors override the influence of the superficial cutaneous receptors (Morelli et al., 1990). Cutaneous receptors, evaluated independently, have been activated by light fingertip pressure and electrical stimulation; both cases resulted in spinal reflex facilitation (Delwaide & Crenna, 1984). To further investigate the influence of cutaneous receptors on spinal excitability, Morelli et al. (1999) applied topical anesthetic to the plantar flexors prior to massage. This was intended to limit the effect of superficial receptors and isolate the effects of massage on deep mechanoreceptors; however, no change in excitability was observed. The basic mechanisms of massage-induced changes in spinal excitability have yet to be identified. It has been proposed that groups of rapidly adapting intrafascial mechanoreceptors are responsible for mediating neural responses during and after tissue manipulation and that the intensity and location of the intervention are important factors in determining the degree of modulation.

1.7 Conclusion

The current literature suggests that FR and RM are an effective means of increasing ROM and altering pain perception in various muscle groups. There is conflicting evidence to support the use of FM and RM as a means of improving athletic performance, although a number of studies have reported increases in physiological performance following acute bouts of FM or RM. Interestingly, the underlying mechanisms for these improvements have yet to be identified. The global nature of these improvements suggests the possibility for neural mechanisms in addition to local muscular changes. Examining the H-reflex during and after rolling will provide an accurate measure of neural responses that may be mediated by FR or RM. Evidence from manual massage therapy suggests that stimulation of mechanoreceptors decreases spinal excitability and that the reduction is intensity-dependent. If the excitability of the neuromuscular system is altered during or after rolling, it is plausible that increases in ROM and pain perception may be due to spinal-mediated changes resulting from increased afferent input in the form of FR or RM.

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Chapter 2: Co-Authorship Statement

The following details my role in the preparation of the manuscript.

Research Design

Methodology was developed based on previous research by Dr. David Behm in combination with work from manual massage therapy. Discussions with Dr. David Behm and Ms. Alyssa-Joy Spence helped to refine details of the experiment. With assistance from Dr. David Behm I was able to obtain approval from the Health Research Ethics Authority (HREA) to conduct this research.

Data Collection

All data was collected by me with assistance from Dr. David Behm and Ms. Alyssa-Joy Spence.

Data Analysis

I performed all data analysis procedures.

Manuscript Preparation

I wrote the manuscript with assistance from Dr. David Behm and Ms. Alyssa-Joy Spence.

Chapter 3: Manuscript

Roller massage decreases spinal excitability to the soleus

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3.1 Abstract

Introduction: Roller massage (RM) interventions have shown acute increases in range of motion (ROM) and pain pressure threshold (PPT). It is unclear whether the RM-induced increases can be attributed to changes in neural or muscle responses. The purpose of this study was to evaluate the effect of altered afferent input via application of RM on spinal excitability, as measured with the Hoffmann (H-) reflex. **Methods:** A randomized within-subjects design was used. Three 30-second bouts of RM were implemented with 30-seconds of rest between bouts. The researcher applied RM to the plantar flexors with a pain perception intensity of 7/10, 4/10, or 0/10 (sham) on a visual analogue pain scale. Measures included full H-reflex/M-wave recruitment curve, H-reflex/M-wave (H/M) ratio, as well as M-wave and H-reflex peak-to-peak amplitudes prior to, during, and up to three-minutes post-intervention. **Results:** RM resulted in significant decreases in soleus H-reflex amplitudes ($p < .001$; $d = 0.87$). High intensity, moderate intensity, and sham conditions decreased soleus H-reflex amplitudes by 58%, 43%, and 19%, respectively. H-reflexes induced with high intensity rolling discomfort or pain were significantly lower than moderate ($p = .045$; $d = .38$) and sham ($p = .011$; $d = .58$) conditions. The effects were transient in nature, with an immediate return to baseline following RM. **Conclusion:** This is the first study to demonstrate that RM-induced increases in afferent input to the spinal cord can inhibit spinal excitability. An intensity-dependent response was observed, indicating that rolling pressure or pain perception may play a role in modulation of the inhibition. Roller massage-induced neural modulation of spinal excitability may explain previously reported increases in ROM and PPT.

Key words: self-massage; neuromuscular rolling; Hoffmann reflex; pain

3.2 Introduction

Foam rolling (FR) and roller massage (RM) are extensively used in exercise and sport practice and are often referred to as forms of self-massage or self-myofascial release. Massage is thought to benefit users through its biomechanical, physiological, neurological, and psychological mechanisms (Weerapong, Hume, & Kolt, 2005); it is plausible that benefits of FR and RM are accrued through similar mechanisms. Many approaches to manual therapy focus their treatment on the fascia through the guided application of manual pressure (Schleip, 2003). Fascia is defined as fibrous collagenous tissues, which are part of a body wide tensional force transmission system (Schleip, Jager, & Klingler, 2012) and is populated with three groups of mechanoreceptors: type Ib Golgi tendon organs, type II Pacini corpuscles and Ruffini endings, and type III/IV interstitial myofascial tissue receptors. The most abundant of all intrafascial mechanoreceptors, type III and IV sensory nerves are comprised primarily of unmyelinated free nerve endings, which are responsive to changes in pain and mechanical tension or pressure (Schleip, 2003). It is believed that FR and RM activate a variety of intrafascial mechanoreceptors resulting in altered afferent input.

Recent evidence highlights the importance of the neurological mechanisms underpinning FR- and RM-induced increases in pain perception and pain pressure threshold (PPT). An increase in pain perception indicates higher levels of pain, while an increase in PPT indicates an increased tolerance to pain. Pain perception was first evaluated by MacDonald et al. (2014), who found an increase in perceived pain during 60-sec of FR. Bradbury-Squires et al. (2015) followed a similar line of investigation and found that 60-sec of RM was painful and induced low levels of muscle activity, likely due to anticipation of the discomfort. Increases in PPT have been noted in both the ipsilateral (Casanova et al., 2017; Jay et al., 2014; Macdonald et al., 2014; Pearcey et al., 2015; Vaughan & McLaughlin, 2014) and contralateral (Aboodarda, Spence, & Button, 2015) limbs. Aboodarda et al. (2015) found a transient and non-local decrease in pain associated with muscle tender points in the plantar flexors (PF). Kelly et al. (2016) reported a FR-induced increase in ROM in both the ipsilateral and contralateral PF and proposed an increase in pain tolerance, similar to the theory of stretch tolerance (Magnusson et al., 1996), as the mechanism responsible. In response to evoked pain, Cavanaugh et al. (2016) found that RM diminished sensations of pain in both the ipsilateral and contralateral PFs. The global nature of these effects suggests the contribution of a central pain-modulatory system that may alter nervous system activity in response to FR- and RM-induced pain.

The Hoffmann (H-) reflex serves as a valuable neurophysiological tool for evaluating the adaptive plasticity of the spinal reflex pathway (Zehr, 2002). While often reported as a measure of motoneurone excitability, the H-reflex is more appropriately used as a measure of presynaptic inhibition at the Ia-alpha motoneurone synapse and to measure the excitability of the neural components of the reflex arc (Misiaszek, 2003). It is well documented that a number of massage therapy techniques and intensities decrease the H-reflex amplitude in the PF (Behm et al., 2013; Goldberg, Sullivan, & Seaborne, 1992; Morelli, Chapman, & Sullivan, 1999; Morelli, Seaborne, & Sullivan, 1990; Morelli, Sullivan, & Chapman, 1998; S. J. Sullivan, Williams, Seaborne, & Morelli, 1991). This can been interpreted as a decrease in spinal excitability, likely due to presynaptic inhibition of Ia afferents (Rudomin & Schmidt, 1999) or a reduction in synaptic efficacy at the Ia afferent-alpha motoneurone synapse (Hultborn et al., 1996), although it is also necessary to consider the contributions of oligosynaptic pathways and reductions in motoneurone excitability (Knikou, 2008; Zehr, 2002). While Morelli and colleagues found that three to four minutes of massage significantly reduced spinal excitability, Behm et al. (2013) reported similar reductions following 30-seconds of massage. Cessation of massage resulted in an immediate return to baseline in all cases, suggesting reductions in spinal excitability are transient in nature.

It appears as though the inhibitory effects of massage are pressure-dependent in addition to transient. Both Behm et al. (2013) and Goldberg et al. (1992) found that deep massage caused a greater reduction in spinal excitability than light massage. To investigate the relative contribution of cutaneous and deep mechanoreceptors, Morelli et al. (1999) applied topical anesthetic to the PF prior to massage. This was intended to limit the effect of superficial cutaneous receptors and evaluate the effects of massage on deep mechanoreceptors. No difference in spinal excitability was found and it was concluded that deep mechanoreceptors likely override the effects of cutaneous receptors during massage. Similarly, Aboodarda et al. (2015) found that heavy RM exerted noxious deep tissue pressure and increased PPT to a larger degree than light RM. Since light RM was not a noxious stimulus, the authors suggested the decrease in PPT with light RM may be associated with increased sensitivity of superficial nociceptors activated via light touch. While the evidence suggests that both light and heavy massage lead to changes in the neurophysiology of underlying tissue, there have been no attempts to determine the mechanisms responsible for mediating such changes.

Therefore, the aim of the present study was to evaluate the neurological mechanisms responsible for changes in ROM following an acute bout of RM. The H-reflex provides a sensitive measure of immediate changes in the spinal reflex pathway that may be mediated by RM. Specifically, we compared the effects of heavy, moderate, and light pressure RM on spinal excitability in the soleus muscle. We hypothesized that RM would immediately alter afferent feedback, resulting in a decreased H-reflex amplitude due to changes in presynaptic inhibition of Ia afferents. A second aim of the study was to assess the time course of acute changes in spinal excitability, using multiple bouts of RM and assessing changes for up to 5-min post-RM. We hypothesized that decreases in H-reflex amplitude would be transient, pressure-dependent.

3.3 Methodology

Participants:

Based on prior related studies (Cavanaugh et al., 2016; Grabow, Young, Alcock, et al., 2017; Grabow, Young, Byrne, Granacher, & Behm, 2017; Halperin, Aboodarda, Button, Andersen, & Behm, 2014), a statistical power analysis indicated that a minimum of 18 participants would be needed to attain an alpha of 0.05 with a power of 0.8. A convenience sample of eighteen healthy individuals (ten males; 25 ± 3 years, 179.1 ± 7.7 cm, 81.5 ± 10.26 kg, and eight females; 24.0 ± 1.77 years, 169.7 ± 8.22 cm and 65.7 ± 8.64 kg) volunteered to participate in this study. All participants reported being recreationally active, engaging in resistance training and/or aerobic exercise at least twice per week. Exclusion criteria included any history of neurological or musculoskeletal injuries in the past year. Participants were instructed to refrain from vigorous physical activity and from consuming alcoholic beverages 24 hours prior to testing. Caffeinated beverages and dietary supplements were not to be consumed within 6 hours prior to testing. All participants were verbally informed of the experimental protocol and gave written informed consent approved by the Newfoundland and Labrador Human Research Ethics Board (reference #2016.076).

Experimental procedures:

The protocol included testing for H-reflex and muscle action potential wave (M-wave) with a number of electrical stimulation intensities. Using a randomized within subjects design, three experimental sessions were conducted on separate testing days with at least 24-hours between sessions. Interventions involved a RM device applied by the researcher to the PF on the dominant leg. Three sets of 30-seconds of rolling was chosen based on prior published work from this laboratory which demonstrated a significant increase in PF ROM (Halperin et al., 2014) and pain pressure threshold (Aboodarda et al., 2015), or diminished evoked pain (Cavanaugh et al., 2016). All rolling was conducted at a cadence of 30 beats per minute. Participants lay prone with the head turned to the right on a padded table for the duration of the experiment. Ambient light was reduced and all participants wore earplugs to minimize competing stimuli.

Interventions:

A Thera-band® RM (Hygienic Corporation, Akron, OH, USA) was used for the duration of the experiment. The RM consisted of a hard rubber material (24 cm in length and 14-cm circumference) with low amplitude, longitudinal grooves surrounding a plastic cylinder (Halperin et al., 2014; Sullivan, Silvey, Button, & Behm, 2013). The RM was applied to the PF at the following three rolling intensities: 7/10 pain, 4/10 pain, and 0/10 pain (sham) on a Visual Analog Scale (VAS). Pain perception has been used extensively to control the intensity of RM interventions (Cavanaugh et al., 2016; Grabow, Young, Alcock et al., 2017; Halperin et al., 2014). The 7/10 and 4/10 intensities were chosen to represent high and moderate rolling pressures and pain levels. The sham condition consisted only of light touch and did not elicit pain. The same researcher applied the RM to the PF for three sets of 30-seconds with 30-seconds rest. Participants were instructed to provide feedback during RM to ensure the appropriate intensity of pain was reached and maintained throughout each of the 30-second bouts.

Electromyography:

The skin surface was prepared by shaving the target area, followed by cleaning and abrading with alcohol swabs to remove dead epithelial cells. Two surface electrodes (1-cm

Ag/AgCl; MediTrace 133, Kendall, Technical products Toronto, Ontario, Canada) with an inter-electrode distance of 2 cm were placed on the soleus, gastrocnemius, and tibialis anterior. The lateral aspect of the soleus, directly below the gastrocnemius-soleus intersection, was chosen to limit the movement artifact associated with RM of the PF. The reference electrode was placed on the lateral malleolus. EMG was sampled at 2500 Hz, filtered with a Blackman 61 dB band-pass filter between 10 and 500 Hz, amplified (bi-polar differential amplifier, input impedance of 2 MOhms, common mode rejection ratio of 110 dB min (50/60 Hz), gain of 1,000, noise of ± 0.05 mV), and stored for further analysis.

INSERT FIGURE ONE APPROXIMATELY HERE

Nerve stimulation:

H-reflex generation required monopolar electrical stimulation of the posterior tibial nerve. An anode (1-cm Ag/AgCl; MediTrace 133, Kendall, Technical products Toronto, Ontario, Canada) was placed superior to the patella, on the anterior thigh. The position of the cathode over the posterior tibial nerve was manipulated using a probe electrode at the beginning of each session to obtain the highest amplitude H-reflex with the lowest intensity stimulation. Once this position was located, the cathode (1-cm Ag/AgCl; MediTrace 133, Kendall, Technical products Toronto, Ontario, Canada) was secured with an elastic bandage and foam ball to apply additional pressure to the nerve. Electrodes were connected to a high-voltage constant-current stimulator (Stimulator Model DS7H+; Digitimer, Welwyn Garden City, Hertfordshire, UK). The amperage (1-1000 mA) of a single square-wave pulse lasting 1-ms was manipulated to achieve desired stimulator output.

At the beginning of each session, recruitment curves of the H-reflexes and M-waves were measured under resting conditions to determine the maximum amplitudes of the H-reflex (H_{max})

and M-wave (M_{max}). Stimulations were increased by 2 milliamps every 10-seconds until the M_{max} was reached. The corresponding intensity for all stimulations was retained for analysis. Following the recruitment profile, a 10-minute break was given. Stimulation intensity was chosen to be the amperage that elicited a value of 50% of the H_{max} of the ascending limb in the recruitment curve because it is sensitive to both facilitation and inhibition (Meinck, 1980). The stimulus intensity was monitored using the corresponding evoked M-wave (5-10% of M_{max}). An evoked M-wave is recommended to monitor the stimulation conditions and to ensure a consistent level of motoneurone pool and muscle membrane action potential activity throughout the experiment (Zehr, 2002).

INSERT FIGURE TWO APPROXIMATELY HERE

Once the appropriate stimulation intensity was chosen, a control trial was given to mimic the intervention and serve as a measure of stimulator output consistency. Stimulations were delivered at the selected intensity every 10-seconds for three-minutes under resting conditions. The control trial was followed by an additional 10-minute break. During the intervention, the researcher applied RM to the PF at a predetermined intensity for three 30-second bouts with 30-seconds rest. Stimulations were delivered at the selected intensity every 10-seconds during the three minute intervention period and continued every 10-seconds for one- and three-minutes immediately following the intervention.

Data acquisition and analysis:

All analog data were digitized using a 12-bit A/D board (Biopac Systems Inc., DA 150: analog-digital converter MP150WSW; Holliston, Massachusetts) and stored on a computer running Acqknowledge 4.1 Software (Biopac Systems Inc., Holliston, Massachusetts). The peak-to-peak amplitudes of each M-wave and H-reflex were determined from the unrectified EMG signals. To assess background activity of the ipsilateral soleus muscle, data was analyzed with a root mean squared moving window of 50-ms and the maximum value noted. Both H-reflex and M-wave peak-to-peak amplitudes were normalized to and expressed as a percentage of the M_{max} attained in each session. Data were grouped and averaged for subsequent analysis.

Statistical analysis:

Statistical analyses were computed using the SPSS software (Version 23.0, SPSS, Inc, Chicago, IL, USA). The assumption of sphericity and normality was tested for all dependent variables. If the assumption of sphericity was violated, the corrected value for non-sphericity with Greenhouse-Geisser epsilon was reported. Intra-session reliability of the pre-test M- and H-responses were assessed with Cronbach's alpha intraclass correlation coefficient (ICC) measurements. A three-way within subjects ANOVA (3x3x2; 3 pain intensities x 3 sets of RM x 2 interventions (RM or Rest); n=18) was used to identify specific main effects and interactions during the RM intervention. To assess the time course of changes in the H-reflex, a second three-way within subjects ANOVA (3x3; n=18) was utilized to determine the effect of the three intensities of pain on the H-reflex at pre-test, 1-min, and 3-min post-intervention. When the F value was significant, a Tukey's HSD test was conducted for post hoc comparison. If significant interaction effects were revealed, paired samples *t*-tests were applied. Cohen's *d* effects sizes (ES)(Cohen, 1988) were also calculated to determine the magnitude of the differences between interventions and time. The following criteria were used: ES < 0.2 was classified as trivial, ES = 0.2–0.49 was considered a "small" effect size; ES = 0.5–0.79 represented a "medium" effect size; and ES > 0.8 represented a "large" effect size. Correlations between pain intensity and background EMG (bEMG) were performed using Spearman's rho and analyzed using the following criteria: ± 0.1 represented a small effect, ± 0.3 represented a medium effect, and ± 0.5

represented a large effect. Significance level was set at $p < .05$. Group data are shown as mean ± the standard error of the mean (SEM).

3.4 Results

A summary of intra-session descriptive and reliability measures is available in Table 1. ICCs for pre-test measures were very reliable for both the M-response (0.996 intra-session) and the H-reflex (0.954 intra-session).

INSERT TABLE ONE APPROXIMATELY HERE

The first three-way within subjects ANOVA (3x3x2; n=18) revealed significant main effects for pain intensity ($F_{(2,34)} = 5.24$, $p = .01$), intervention ($F_{(1,17)} = 65.919$, $p < .001$), and interaction effects for pain intensity and intervention ($F_{(2,34)} = 9.306$, $p = .001$). Pairwise comparisons revealed that RM-induced high intensity pain significantly reduced H-reflex amplitudes relative to moderate intensity ($p = .045$; $d = .38$) and sham ($p = .011$; $d = .58$) conditions. Moderate intensity pain was not significantly higher than sham ($p = .294$; $d = .27$). RM reduced H-reflex amplitude by 39% with a large effect across all conditions, independent of pain intensity ($p < .001$; $d = .87$). Paired samples T-tests revealed significant decreases in H-reflex amplitude by 58 % with a large effect during high intensity ($t_{(17)} = -6.068$, $p < .001$; $d = 1.12$), 43 % with a large effect during moderate intensity ($t_{(17)} = -6.129$, $p < .001$; $d = .95$), and 19 % with a medium effect during sham ($t_{(17)} = -6.198$, $p < .001$; $d = .48$) conditions (see Figure 3).

INSERT FIGURE THREE APPROXIMATELY HERE

The second two-way within subjects ANOVA (3x3; n=18) did not reveal any main effects for pain intensity ($F_{(2,34)} = .002$, $p = .998$) or time ($F_{(2.27,38.59)} = 2.061$, $p = .143$). The amplitude of the H-reflex returned to baseline immediately following the removal of the RM (see

Figure 4) and there were no significant differences observed between the pre- and post-intervention periods at any of the three pain intensities.

INSERT FIGURE FOUR APPROXIMATELY HERE

A bivariate correlation was calculated between bEMG and pain intensity. Spearman's rho correlation coefficient was chosen because bEMG data were not normally distributed according to the Kolmogorov-Smirnov normality test (Soleus $D_{(51)} = 0.283$, $p < .001$; Gastrocnemius $D_{(51)} = 0.292$, $p < .001$; Tibialis Anterior $D_{(51)} = 0.278$, $p < .001$). These variables were significantly, positively and highly correlated (Soleus $\rho = .64$, $p < .001$; Gastrocnemius $\rho = .64$, $p < .001$; Tibialis Anterior $\rho = .78$, $p < .001$) indicating that there was a positive relationship between the intensity of pain and the level of bEMG.

3.5 Discussion

The most important finding of the present study was that 30-sec of RM significantly reduced H-reflex amplitudes at all three intensities. Additionally, the reduction in H-reflex amplitude was intensity-dependent, with a greater relative reduction during the high and moderate intensity conditions relative to sham. These effects were transient in nature, with an immediate return to baseline across all three conditions; no residual effects were observed for up to three-min post-intervention. Finally, a large positive correlation between intensity of pain and level of bEMG was observed.

A reduction in H-reflex amplitude following massage is not without precedent; however, this is the first evidence of RM-induced modulation of spinal excitability. Similar to findings from manual massage of the PF, a marked reduction in spinal excitability was observed during the RM intervention. Morelli et al. (1990) first reported a 71% decrease in H-reflex amplitude during 5-min of manual massage of the ipsilateral PF. Goldberg et al. (1992) later reported a difference between deep and light massage, noting a 49% and 39% reduction in amplitude during massage, respectively. Additionally, Behm et al. (2013) found an 85% and 57% reduction in the soleus H/M ratio following vigorous muscle tapping and light massage, respectively. The greater inhibition of the deep or vigorous massage suggests that deep mechanoreceptors override the influence of the more superficial cutaneous receptors (Goldberg et al., 1992). The results of the present study further support an intensity-dependent reduction in spinal excitability during RM as we observed a reduction in H-reflex amplitude of 58%, 43%, and 19% in the high, moderate, and sham conditions, respectively.

Recent work from our lab used a constant pressure device in addition to pain perception to assess changes in ROM following different intensities of RM (Grabow, Young, Alcock, et al., 2017). Changes in ROM and subsequent performance were not intensity-dependent, suggesting that high levels of RM-induced pressure and/or pain are not necessary for increasing ROM. The results of the present study suggest that an increase in RM-induced pressure and/or pain resulted in a more profound decrease in spinal excitability, although a noxious stimulus was not required to obtain a statistically significant degree of H-reflex inhibition (see Figures 3 and 4). Similarly, Grabow et al. (2017) demonstrated that discomfort was not required to observe increases in ROM following RM. It is possible that a curvilinear relationship exists between RM-induced inhibition at the spinal level and increased ROM. That is, while higher intensity RM induces greater reduction in spinal excitability, the subsequent change in ROM may plateau or display a ceiling effect, as shown by Grabow et al. (2017). It is possible that factors other than reflex inhibition, such as changes in cortical motoneurone excitability (Misiaszek, 2003), viscoelastic properties or fascicle pennation angles (Casanova et al., 2017) may also be involved in mediating improvements in ROM following RM. It is important to note that while reductions in spinal excitability following a variety of massage therapy techniques have been documented (Behm et al., 2013; Goldberg et al., 1992; Morelli et al., 1998), this is the first study to report the inclusion of a noxious, or painful, stimulus.

Due to the pain associated with moderate to high intensity RM, Aboodarda et al. (2015) proposed that the ascending and/or descending central pain-modulatory systems are activated during RM. Descending control of spinal excitability is well documented (Bannister & Dickenson, 2017). It is likely that RM stimulates a variety of mechanoreceptors and nociceptors, which can alter the transmission of ascending nociceptive information via small diameter Aδ fibres and give rise to a descending inhibitory effect that allows for modulation of spinal excitability (Melzack & Wall, 1965; Moayedi & Davis, 2013). Additionally, Mense (2000) highlights the importance of descending spinal pathways, which can modulate pain sensations by either inhibiting or facilitating the discharge of spinal nociceptive neurones. These descending pathways, triggered by noxious stimuli, have widespread terminations in the spinal cord (Le Bars, Villanueva, Willer, & Bouhassira, 1991) and may explain the reduction in spinal excitability observed in the present study.

A decrease in spinal excitability could be due to an increase in inhibition or a decrease in facilitation, though it is often challenging to distinguish where the exact change occurs. The most plausible explanation for the observed decrease in spinal excitability can be attributed to presynaptic inhibition of Ia afferents due to activation of mechanoreceptors and nociceptors in the skin, muscle, and fascia (Behm et al., 2013). As hypothesized, higher intensities of RM-induced pressure and pain provided a stronger stimulus, increasing afferent input and thereby increasing levels of presynaptic inhibition, resulting in larger reductions in spinal excitability. It is unlikely that substantial change in viscoelastic properties or mechanical deformation of tissues is possible during such short duration or moderate intensity RM (Schleip, 2003). Chaudhry et al. (2008) demonstrated that forces beyond normal physiological ranges are required to produce compression and shear in the plantar fascia. These findings support the notion that the benefits of RM may be accrued through neural responses rather than changes in tissue compliance and the musculotendinous unit (Macdonald et al., 2014). The transient nature of these effects suggests a group of highly sensitive, rapidly adapting receptors is responsible for mediating these responses.

The significant positive correlation between levels of bEMG and the intensity of pain indicates an increase in neural activity to the lower limb during the RM intervention. Bradbury-Squires et al. (2015) found that 60-sec of roller massage induced pain in both the biceps femoris and vastus lateralis, evidenced by increased EMG activity upwards of 8% of maximal activation. Co-contraction of musculature in anticipation of discomfort is a natural response (Pijnappels, Bobbert, & van Dieen, 2006) and the current findings suggest that the level of co-contraction is dependent on the level of anticipated discomfort. Interestingly, the strongest correlation between bEMG and intensity of pain was found in the TA muscle. It is possible that reciprocal inhibition of the soleus motoneurones via increased TA activity could explain the decrease in spinal excitability to the soleus (Crone, Hultborn, Jespersen, & Nielsen, 1987); however, this would not explain the sham condition, in which all muscles remained electromyographically silent for the duration of the rolling intervention. The results of the sham condition provide strong evidence of presynaptic inhibition of Ia afferents due to increased afferent input via RM application and it is unlikely that changes in motoneurone excitability or reciprocal inhibition are involved.

There are several limitations of the present study that must be considered when interpreting the findings. First, reflex recordings were not conducted during a low level contraction, as is typically seen in the literature (Knikou, 2008; Zehr, 2002). When reflex recordings are taken at rest the relative state of depolarization of the motoneurone pool is not known (Knikou, 2008; Zehr, 2002). Motoneurone pool activation can be estimated by measuring and controlling the background level of muscle contraction, as it is has been shown that the amplitude of the H-reflex will increase with the number of motoneurones recruited in the target motoneurone pool (Schieppati, 1987). The increased levels of bEMG in the current study suggest that the target muscle may not have been activated to the same relative level during each of the RM conditions. RM is a passive process during which there is no voluntary activation of the target muscle; therefore, as a practical consideration it was necessary to take recordings at rest.

Second, if painful RM can induce low levels of muscle activity, the changes in H-reflex amplitude may be caused by activity-dependent axonal hyperpolarization. While the absolute EMG amplitudes in the present experiment are low, no maximal contractions were performed for normalization procedures to allow for interpretation of the relative state of muscle activity. It would be necessary to include a control condition in which the amplitude of the voluntary bEMG is matched to that elicited during painful RM. Therefore, it is possible that changes in background muscle activity may explain some of the observed findings.

Finally, a pain scale was used to quantify the amount of pressure exerted on the participant’s lower leg. Pain is highly subjective and the interaction between nociceptive input and pain perception is complex (Wiech, Ploner, & Tracey, 2008). Consequently, it is likely that the amount of RM-induced pressure varied greatly between participants. The verbal cues given to the participants to describe moderate (4/10) and high (7/10) intensities of pain were “uncomfortable” and “painful”, respectively; therefore, despite a difference in pressure, the inclusion of pain perception to monitor RM-intensity ensured that each participant experienced two considerably different nociceptive stimuli during the moderate and high intensity pain experimental sessions, regardless of subjective differences in pain tolerance.

3.6 Perspectives

The results of the present study provide strong evidence for neural modulation of spinal excitability to the soleus during RM. It remains unclear whether a reduction in spinal excitability to the contralateral limb during RM would occur; however, we speculate that the altered afferent input was not specific to the location and may explain non-local increases in ROM and PPT. These results are of particular importance to athletes and practitioners, who must consider their current rolling practices and recognize the transient nature of the observed reflex inhibition. Future research should investigate the effect of pairing brief, repeated bouts of FR or RM with static stretching, in an effort to capitalize on the reflex inhibition and further improve ROM. Additionally, research on clinical populations, specifically those presenting with myofascial pain, merits inclusion in future investigations.

3.7 Conclusion

This is the first study to attempt to determine the underlying mechanisms responsible for increases in ROM and PPT following RM. RM induces change in afferent input to the spinal cord, resulting in immediate reflex inhibition, likely due to presynaptic inhibition at the Ia-motoneurone synapse. This inhibition is intensity-dependent, with a greater percent reduction during the high and moderate intensity RM conditions, relative to sham. It is plausible that immediate increases in ROM and PPT following brief application of RM can be attributed to the observed inhibition at the spinal level. The transient nature of these effects suggests a group of rapidly adapting receptors is responsible for mediating changes in spinal excitability.

Acknowledgments

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Conflict of interest

There are no conflicts of interest of the authors with the information contained within the manuscript.

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3.9 Tables

Table 1. Intra-session descriptive and reliability measures. RM Int = roller massage intensity, SD = standard deviation, ICC = intraclass correlation coefficient.

M-Wave		
RM Int	**Mean (SD)**	**Intra-session ICC**
High	8.46 (4.15)	0.992
Moderate	8.55 (5.27)	0.998
Sham	7.75 (5.01)	0.999
H-Reflex		
RM Int	**Mean (SD)**	**Intra-session ICC**
High	45.1 (18.60)	0.947
Moderate	44.0 (18.49)	0.980
Sham	43.3 (14.48)	0.934

3.10 Figures

Figure 1. Experimental set-up and location of EMG and posterior tibial nerve stimulating electrodes. RM of the plantar flexors was performed in between the soleus and gastrocnemius electrodes.

Figure 2. Representative (n=1) stimulus recruitment curve. Stimulations delivered every 10-sec in increments of 2mA using a high-voltage constant current stimulator with a single square wave pulse lasting 1-ms. P-P = Peak to peak, mV = millivolt, mA = milliamp.

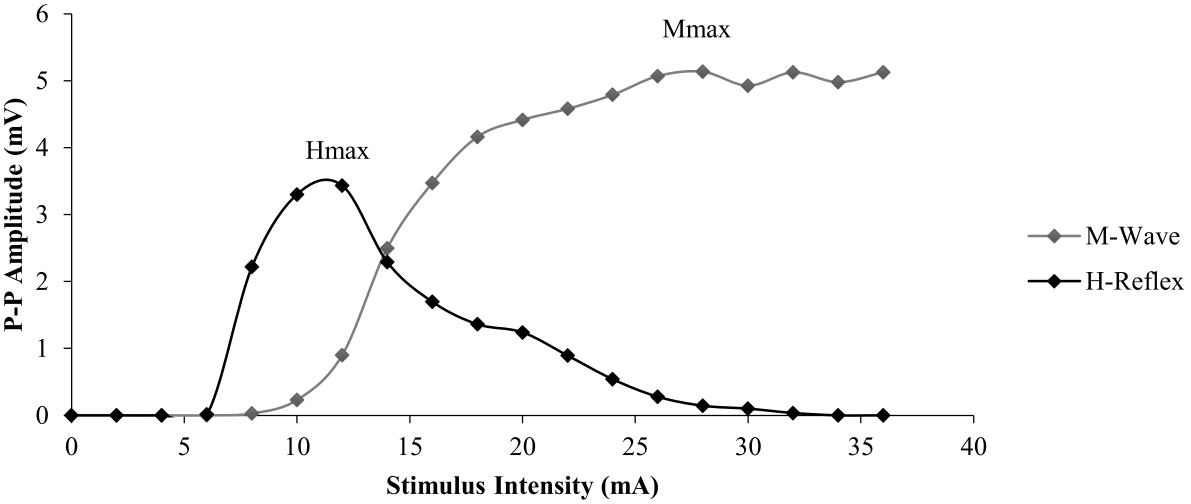

Figure 3. Representative raw data traces (n=1) during three different intensities of RM. **A**: High intensity (7/10) roller massage condition; 49% decrease in H-reflex amplitude during RM. **B**: Moderate intensity (4/10) roller massage condition; 44% decrease in H-reflex amplitude during RM. **C**: Sham (0/10) roller massage condition; 14% decrease in H-reflex amplitude during RM. RM = Roller massage, OFF = Rest period.

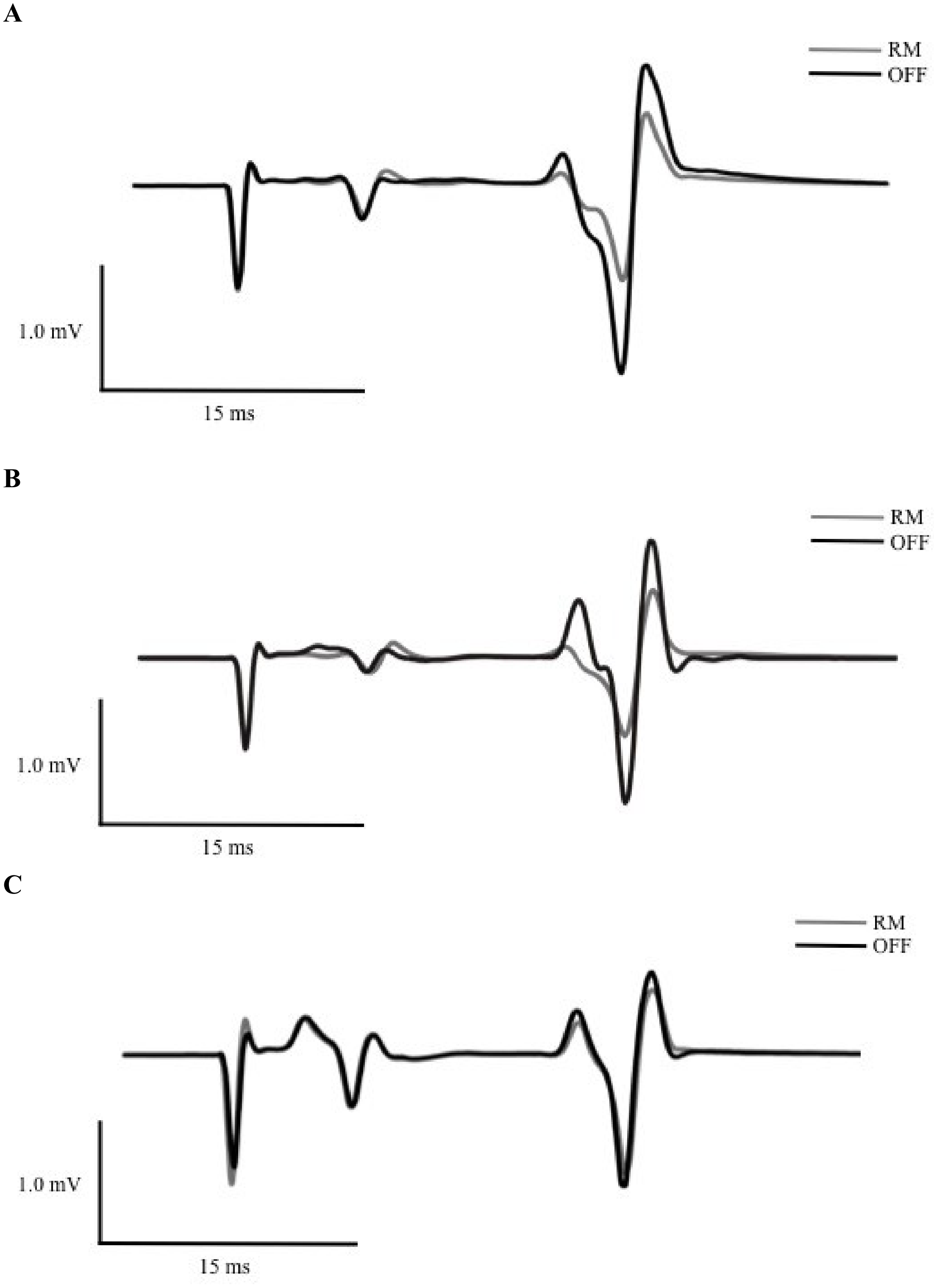

Figure 4. Grouped reflex responses during three roller massage intensities. **A**: High intensity (7/10) roller massage condition. **B**: Moderate intensity (4/10) roller massage condition. **C**: Sham (0/10) roller massage condition. Roller massage applied during stimulations 1-3, 7-9, 13-15. There were significant main effects for H-reflex inhibition with all pain intensities during rolling (1-3, 7-9, 13-15) compared to the rest periods (4-6, 10-12, 16-18).