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REVIEW ARTICLE

Do Self-Myofascial Release Devices Release Myofascia? Rolling Mechanisms: A Narrative Review

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Abstract

The term "self-myofascial release" is ubiquitous in the rehabilitation and training literature and purports that the use of foam rollers and other similar devices release myofascial constrictions accumulated from scar tissue, ischaemia-induced muscle spasms and other pathologies. Myofascial tone can be modulated with rollers by changes in thixotropic properties, blood flow, and fascial hydration affecting tissue stiffness. While rollers are commonly used as a treatment for myofascial trigger points, the identification of trigger points is reported to not be highly reliable. Rolling mechanisms underlying their effect on pain suppression are not well elucidated. Other rolling-induced mechanisms to increase range of motion or reduce pain include the activation of cutaneous and fascial mechanoreceptors and interstitial type III and IV afferents that modulate sympathetic/parasympathetic activation as well as the activation of global pain modulatory systems and reflex-induced reductions in muscle and myofascial tone. This review submits that there is insufficient evidence to support that the primary mechanisms underlying rolling and other similar devices are the release of myofascial restrictions and thus the term "self-myofascial release" devices is misleading.

Key Points

Rolling is often described as a self-myofascial release technique in the literature but the term may be misleading.

While rolling can affect the myofascial tissues, other mechanisms such as rolling-induced global (full body) effects on muscle tone from changes in the parasympathetic system, reflexes and pain tolerance may have even greater influence on improvements in range of motion and pain sensitivity.

1 Introduction

The use of devices such as foam rollers, roller massagers, balls and other instruments that purportedly increase range or motion (ROM), decrease myofascial pain, improve recovery from exercise-induced muscle damage and improve performance has experienced a recent surge in the exercise science, physical therapy and rehabilitation literature [1–5]. Although the magnitude is highly variable, ranging from 3 to 23% [6, 7], rolling generally increases ROM in the short-term [6, 8–14] for up to 20 min [15–17]. Increases in joint ROM have been reported with as little as 5–10 s of rolling [14], but the vast majority of research implements multiple sets of 30–60 s of rolling. While the rolling durations have not been directly compared, the results tend to indicate that 60 s of rolling provides more enhanced ROM [9–13]. However, in one study, 60, 90 and 120 s of rolling were applied between four sets of knee extensions. While 120 s of rolling decreased the number of knee extension repetitions by 14%, the 90 and 60 s of rolling also decreased repetition numbers by 8–9% [18]. This was one of the few studies that reported impairments with rolling. Regarding the persistence of rolling effects, Hodgson et al. [19] showed that following a typical warm-up (aerobic activity, static and dynamic stretching, and sport-specific activities), when rolling was performed at

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10-min intervals, the increased ROM persisted for 30 min to a greater degree than without intermittent rolling.

Whereas static stretching (SS) also improves ROM, a major concern is that prolonged SS application can lead to performance deficits [20–22]. An advantage of rolling is that it does not typically impair subsequent muscle strength [12, 14, 23–29], jump height [23, 28, 30, 31], sprint time [32], and fatigue endurance [33]. In some studies, rolling actually was shown to improve strength [10, 34], power [34], sprint speed [34], neuromuscular efficiency during a lunge [9], and knee joint proprioception [35]. A recent meta-analysis determined that the effects of foam rolling on performance and recovery are generally small to negligible, with some exceptions such as improved sprint performance and flexibility or muscle pain reduction [5].

1.1 Rolling Devices

While a variety of rolling devices have been utilized in the research (i.e. foam rollers, roller massagers, balls), there have not been any studies that have directly compared their effectiveness. The following information presented in this review illustrates that rolling effects of foam rollers and roller massagers upon ROM and performance (i.e. strength and power) measures seem to be relatively similar. The architecture of the roller has been examined in two studies. Cheatham and Stull [36] compared moderately firm multilevel and grid pattern foam rollers to a smooth roller and found that the smooth roller provided less positive changes in ROM and pain-pressure thresholds. Curran et al. [37] reported that a multi-level rigid foam roller exerted higher pressures over smaller contact areas than a smooth roller, suggesting that the multilevel roller would have greater benefits with self-myofascial release. However, whether the roller has multi-levels or is rigid or softer, is it possible that these devices can release myofascia and what does that term mean?

1.2 Self-Myofascial Release: A False Friend?

In many instances, the improved ROM and performance measures are ascribed to the effect of rollers releasing myofascial constrictions due to scar tissue, ischaemia-induced muscle spasms and other pathologies [38, 39]. Thus, a term often used to describe the action of these tools (rollers, balls and others) is self-myofascial release techniques or devices. A search of the term *self-myofascial release* on titles and abstracts within the database PubMed posts 25 hits since 2005, with most hits post-2013. When the terms *foam roller* and *roller massage* are searched from the titles and abstracts within PubMed, 48 hits are observed from 2005–2019, again with most occurrences within the last 6 years. Hence, self-myofascial release is associated with the effects of rolling in a substantial portion of the published literature. While the term self-myofascial release is often used in the literature and by practitioners, evidence that myofascial release is a predominant mechanism permitting the augmentation of flexibility, pain suppression or performance enhancement is quite scant. To the uninformed, the term self-myofascial release may perpetuate an incorrect or false mechanism of rolling. Hence, it is important to evaluate rolling mechanisms and correct any misconceptions, and thus enlighten researchers, health and fitness professionals, and practitioners about the mechanisms underlying rolling. It is the purpose of this narrative review to survey the literature and examine whether self-myofascial release is a legitimate term to describe these roller devices and their actions and to highlight alternative mechanisms.

2 Myofascial Tone and Soft Tissue Restrictions

Fascial restrictions are reported to occur in response to inactivity, overload, injury, inflammation and disease. Several mechanisms, acting individually or in concert, may trigger stiffness increases of the connective tissue: (1) sustained volume changes of the muscle, e.g. following hypertrophy or hypertonicity, affect the fascia's mechanical properties due to the radial expansion of the muscle [40]. (2) In some areas, skeletal muscles present direct fiber insertions into the surrounding deep fascia, which allow for selective tensioning [41]. (3) Contraction of myofibroblast cells, whose activity is governed by the autonomic nervous system, can lead to long-term increases of fascial tone, particularly in the presence of psychological stress [42]. (4) Finally, fascial hydration changes occur in response to mechanical stimuli such as stretching exercises. This is of importance because alterations in the water content are directly related to connective tissue stiffness [43].

In addition to general tissue stiffening, myofascial trigger points (MTrPs) represent a special pathology, which has been related to the connective tissue. When fascia becomes dehydrated, diminishing its elasticity, myofascia can bind around the injured regions, leading to fibrous adhesions [38, 39]. These adhesions may induce "hypersensitive tender spots" [44], also known as trigger points. A MTrP is described as the most irritable location in a taut band of muscle, sensitizing nociceptors in the area [45, 46]. Fibrous adhesions can be painful, prevent healthy muscle mechanics (i.e. altered muscle strength and activation, endurance and coordination) and decrease soft-tissue extensibility impairing joint ROM and muscle length [38, 39, 44].

3 Effects of Alleged Self-Myofascial Release Treatments on Trigger Point Pain

Generally, a classical treatment of MTrP is to apply manual pressure to the location. According to Lucas et al. [47], other treatments include injections, needling, spray, stretch, and massage; however, the evidence as of 2009 did not show that these treatments were more effective than placebo. They further indicated that the reliability of identifying trigger points was quite variable (weak) and further investigation into trigger points was needed. Similarly, Tough et al. [48] indicated that there was limited consensus on MTrP definition, with more research necessary to test the reliability and validity of the diagnosis, and henceforth claims for effective treatment interventions should be viewed with caution. However, by 2017, Rozenfeld [49] reported that the reliability of detecting trigger points in the hip and thigh area ranged from moderate to substantial. Grieve [50] screened for active ankle dorsiflexion restriction and the presence of latent trigger points, and treated with a combination of manual pressure release and 10-s passive stretch. They reported a statistically significant, clinically meaningful and large magnitude increase in ankle ROM.

There is a paucity of research elucidating the effect of using "self-myofascial release" devices in the treatment of trigger points. Grieve et al. [51] employed a multimodal intervention using therapist and self-applied trigger point pressure release as well as stretching and reported improvements in lower extremity functional scale 6 weeks post-treatment. However, due to the lack of a control group and the combination of different treatments, no assumptions on the relative impact of self-release can be drawn. Wilke et al. [52] examined static compression and dynamic foam rolling over MTrP in comparison with a placebo condition. Static compression, indeed, was the only treatment reducing pain. Yet, presumably due to the small sample, the difference to the placebo control did not reach statistical significance. In summary, although signs indicate that self-massage with rolling devices may reduce MTrP sensitivity, more research is needed in order to definitely substantiate this claim. More importantly, although, manual pressure release seems to improve function and possibly pain, the question arises as to whether these effects actually stem from the release of myofascial adhesions, scar tissue or taut bands.

4 Does Myofascial Tissue Release Following Self-Massage?

While the aforementioned Grieve and colleague studies as well as others have reported improved function and decreased pain with manual therapy using the therapists' fingers, hands and elbows; are these manipulations actually releasing general tissue stiffness, adhesions and trigger points or are there other mechanisms at work? Basically, two pathways for the modification of soft tissue stiffness can be distinguished.

On the one hand, the central nervous system is a powerful moderator of muscle tone. If the neural drive to the active component of the locomotor system is reduced, this may lead to altered hardness of the entire myofascial system. Autogenic inhibition is based on the sensory feedback of the Golgi tendon organ [53, 54]. Registering increases in tension, its stimulation potentially leads to a reduction in muscle activity. Both the reflex as well as a reduction in stretch tolerance may explain increases in ROM following flexibility exercises [20–22].

On the other hand, peripheral changes of the mechanical tissue properties (arguably rather than in the collagenous connective tissue) may occur, achieving a similar effect. As outlined above, exercise can substantially affect the water content but also other circulatory parameters such as blood flow. In addition, exercise has been shown to have an influence on viscoelasticity of biological tissues.

According to our analysis of the literature, there are reasons for and against the theory that tool-assisted soft tissue mobilization can achieve a release. The most important possible mechanisms are detailed below.

4.1 Evidence for Direct Effects of Myofascial Release

In recent years, mechanistic studies have mostly examined foam rolling techniques. Wilke et al. [55] used a semi-electronic tissue compliance meter to assess the compressive stiffness of the anterior thigh before and after four 45-s bouts of foam rolling. The treatment induced stiffness decreases ranging between 15 and 24%, which were larger at 10 min post-intervention than immediately after the intervention. Morales-Artacho et al. [56] employed shear-wave elastography to estimate the viscoelastic behaviour of the muscle-fascia complex. Immediately after rolling, a small magnitude (effect size: 0.21) decrease of the elastic modulus (a measure of an object's resistance to non-permanent deformation) was observed. Heiss et al. [57] evaluated the mechanical tissue properties before and after foam rolling of the thigh, using the above-stated protocol (4 × 45 s) of Wilke et al. While the muscular tissue

remained unaffected, acoustic radiation force imaging revealed a significant stiffness reduction (− 13%) of the iliotibial band at 30 min post-treatment in participants with foam rolling experience. These findings suggest that foam rolling can affect tissue stiffness but that the effects seem to manifest with some delay rather than immediately post-intervention.

Other studies have focussed on the circulatory response to foam rolling exercise. Hotfiel et al. [58] used spectral Doppler and power Doppler ultrasound to examine blood flow after an acute exercise bout in the lateral thigh. Post-treatment, the authors observed a 74% increase in peak flow, suggesting substantially enhanced arterial perfusion. Interestingly, even after 30 min, a higher blood flow was still present. The circulatory effect of foam rolling matches with data from Okamoto et al. [59], who demonstrated that a single rolling exercise session targeting the lower leg muscles and the trapezius positively impacts vascular function: 30 min post-rolling, brachial-ankle pulse-wave velocity decreased and plasma nitric oxide concentration increased, indicating a reduction in arterial stiffness and improved vascular endothelial function. When interpreting the findings of Hotfiel and colleagues as well as those of Okamoto et al., it is crucial to note that their outcomes were not tissue-specific and referred to acute local adaptations in the muscle and the connective tissue. However, the fascia has a rich vascular network with all types of blood vessels including arterioles, capillaries and venules [60] and, hence, it is tenable to assume the observed effects to stem from both the muscle and its fascia.

4.2 Evidence Against Direct Effects on Myofascial Release

Schleip [38, 39] suggests the force necessary to break up or remove myofascial adhesions would exceed the physiological limitations of most people. However, a therapist with substantial mass placing pressure on myofascia with their elbow, for example, would impart quite substantial pressures. In contrast, a partial body mass moving over the broad surface of a foam roller or the force applied with the arms on a relatively long roller massager would not achieve similar pressures, and thus would be unlikely to provide sufficient pressures to break up myofascial adhesions.

Strong evidence for alternative mechanisms underlying the inhibition of muscle tender spot pain is provided by crossover or non-local rolling effects. Three studies have illustrated decreases in muscle pain and evoked stimulation-induced pain in the contralateral untreated leg. Aboodarda et al. [8] identified the most sensitive plantar flexors' muscle tender point and then the painful calf was either massaged by a therapist, roller massaged or control (no treatment). A unique fourth condition was the rolling of the contralateral calf. Whereas both manual massage and rolling the affected calf decreased muscle tender point pain sensitivity, roller massage of the contralateral calf significantly reduced the pain sensitivity as well. Cavanaugh and colleagues [61] induced substantial pain by electrically stimulating the tibial nerve of the plantar flexors with maximal and submaximal (70% of maximal current) intensity, high frequency (50 Hz) tetanic stimulation. Once again, rolling the tetanized or contralateral (no stimulation) calf diminished the pain sensitivity. In addition, Cheatham et al. [62] performed foam rolling of the quadriceps and found increased pain pressure thresholds (decreased pain sensitivity) with the ipsilateral hamstrings and contralateral quadriceps. Therefore, without treating or touching the affected muscle (no possibility of mechanical effects), rolling the contralateral muscle decreased pain in three studies, suggesting a global pain modulatory response.

The proposed global pain modulatory response might be related to the gate control theory of pain [63, 64], diffuse noxious inhibitory control (DNIC) [65] or parasympathetic nervous system alterations. The gate control theory involves activation of thick myelinated ergoreceptor (group III and IV afferents) nerve fibers (via activation of percutaneous and muscle mechanoreceptors, metaboreceptors and proprioceptors) that modify the signals from ascending nociceptors via small-diameter Aδ fibers to the periaqueductal grey nucleus [64]. Analgesia arises from descending signals to opioid receptors that inhibit pain with serotonergic and noradrenergic neurons [66]. DNIC is activated by nociceptive stimuli from a distant or non-local tissue. With DNIC, activation of non-local receptors is transmitted to multireceptive, wide dynamic-range convergent neurons in the cortical subnucleus reticularis dorsalis, where it inhibits pain transmission monoaminergically (i.e. norepinephrine and serotonin), reducing pain perception at the affected as well as distant or non-local sites [65–67]. Massage can also activate the parasympathetic system, which acts globally on the body. Parasympathetic stimulation alters serotonin, cortisol, endorphin, and oxytocin levels, diminishing pain perception [44]. Furthermore, a reduction of parasympathetic reflexes could decrease pain sensitivity by reducing myofascial tissue stress by relaxing the strain on the smooth muscles in the soft tissue.

As decreases in pain not only occur in the treated but also in non-local body regions, it could be hypothesized that rolling-induced increases in ROM similarly represent the result of a predominantly neural adaptation. Recent studies have therefore examined the effect of "release"—interventions on flexibility of remote joints. In fact, enhanced ROM has been observed with rolling of the plantar fascia improving hamstrings flexibility [68], rolling of the ipsilateral plantar flexors increasing contralateral ankle dorsiflexion ROM [16], and rolling of the hamstrings increasing shoulder ROM [69].

Despite these intriguing findings pointing towards a significant global effect, only one study has directly compared the targeted and contralateral joint. Applying a rolling treatment for the calf muscles, Kelly and Beardsley [16] reported greater and longer-lasting local ROM increases (2.2–5.9%). However, possibly due to a lack of statistical power (13 participants per group), the between-group differences to the contralateral joint were not significant. The question as to whether local and non-local ROM increases after rolling are different can hence not be answered conclusively.

Irrespective of the above research deficit, increased ROM of the stretched or distant muscles and joints following stretching has been attributed to an increased stretch tolerance [70]. Many individuals and studies have used stretching to the point of maximal or near-maximal discomfort [20–22]. Either due to psychological accommodation (individual becomes accustomed to the level of discomfort) or the aforementioned gate control theory and DNIC pain suppression mechanisms, exposure to uncomfortable or painful stimuli such as high intensity stretching or rolling can increase pain or stretch tolerance globally. However, the stretching or rolling does not need to be unduly painful to increase stretch tolerance. With the Aboodarda et al. study [8], pain depression occurred after light rolling massage. Grabow et al. [27] found that the intensity of rolling (50%, 70% or 90% of the maximum point of discomfort) did not differentially affect the ROM. Nociceptors (pain receptors) are present in both muscle and skin [71, 72], and thus even light rolling can increase the sensitivity of superficial nociceptors. Again, to emphasize the point, non-local increases in ROM or pain thresholds cannot involve a mechanical release of myofascial restrictions or trigger points.

5 Rolling Mechanisms

If the primary mechanism for improving ROM and decreasing pain with rolling is not self-myofascial release that breaks up fascial adhesions, scar tissue or trigger points, then what are the probable mechanisms? Muscle, fascia and skin are highly innervated by sensory neurons [38, 39]. Within the skin layers, Merkel receptors, Meissner corpuscles, Ruffini cylinders and pacinian corpuscles (mechanoreceptors) possess a spectrum of receptor field areas, which respond slowly or rapidly to different stimulation frequencies. Merkel disks (small receptor field) and Ruffini cylinders (large receptor field) adapt slowly and continue to respond as long as the stimulus is present, whereas the Meissner (small receptor field) and Pacinian (large receptor field) corpuscles adapt rapidly and respond to stimulation with a burst of firing activity at the start and cessation of stimulation. Their major responsibilities are for proprioception, and thus would not play a major role in neural inhibition; however, Ruffini and Pacinian receptors may be able to induce muscle relaxation by inhibiting sympathetic activity [73]. Ruffini receptors are more sensitive to tangential forces and lateral stretch [74], which would be prominent with rolling. Hence, it might be surmised that the four mechanoreceptors should respond to slow rolling or massage with Ruffini cylinders and Pacinian corpuscles also responding to high-frequency vibrations (i.e. vibrating foam rollers). However, with no improvement in ROM and a lack of significant difference in rolling-induced muscle stiffness (overall decrease at both rolling speeds) between slow and fast rolling (Wilke et al. 2019), it seems that there may not have been substantially different contributions from the mechanoreceptors. The advantage of higher frequency vibrating foam rollers over non-vibrating foam rollers with knee [75] and hip [76, 77] ROM and greater pain pressure thresholds (decreased pain sensitivity) [75, 77] may provide some evidence for greater mechanoreceptor contributions at higher frequencies. Perhaps the increased global (full body) relaxation, decreases in heart rate and blood pressure with massage contribute to the non-local rolling effects, and can be partially attributed to the manual stimulation of Ruffini and Pacinian receptors [44].

Interstitial type III and IV receptors can also affect sympathetic and parasympathetic activation. These receptors have both low and high threshold sensory capabilities and respond to both rapid and sustained pressure [22]. These multi-modal receptors respond to pain but also serve as mechanoreceptors activated by tension and pressure. They can help modulate decreases in heart rate, blood pressure and ventilation, and lead to vasodilation [78], which can lead to a more relaxed muscle. Again, as they affect global sympathetic and parasympathetic responses, they could influence the ROM, pain sensitivity and performance of distant non-rolled muscles.

Local mechanisms (factors affecting the muscle or myofascia that is rolled) would involve thixotropic effects that would contribute to the increased ROM of the rolled muscle. Thixotropy occurs when viscous (thicker) fluids become less viscous or more fluid-like when agitated, sheared or stressed [79]. Rolling places direct and sweeping pressure on the skin, fascia and muscle inducing friction. The elevated friction-related tissue temperature and the shearing stress from rolling can decrease intracellular and extracellular fluid viscosity, providing less resistance to movement [22]. The thixotropic effects would not apply to global, non-local effects of rolling.

Massage can reduce the afferent excitability of the alpha motoneurons, which can be monitored with the Hoffman (*H*-) reflex. Manual massage [80–82] as well as roller massage [83] have attenuated the *H*-reflex by 40–90%. A diminution of the *H*-reflex may be attributed to decreased alpha

motoneuron excitability or increased pre-synaptic inhibition, reducing the reflex-induced activation of the rolled muscle.

Golgi tendon organs (GTO) (type Ib afferents) respond to musculotendinous tension and strong stretch usually resulting in inhibition. Huang et al. [84] demonstrated that using a single short-duration (10- or 30-s) massage at the hamstrings musculotendinous junction increased ROM without an increase in passive muscle tension or EMG activity. While it is possible that force applied to the tendon would activate the GTO inhibitory responses leading to greater muscle relaxation or decreased tonus, the GTO effects persist for only approximately 60 ms after cessation of stress [85]. Thus, the respective 5.8–11.3% ROM increases immediately after the massage in the Huang et al. study were likely not due to GTO inhibition.

6 Methodological Considerations and Future Research

Although researchers have made considerable efforts to elucidate the effects of rolling treatments, the mechanisms and their relative contributions are still a matter of debate. Future studies should particularly address the following key aspects. Firstly, while some evidence supporting the theories of a mechanistic-structural as well as a neural genesis of rolling-induced effects is available, there is a paucity of trials jointly examining both. It would thus be intriguing to assess local (treated joint) and non-local (non-treated joint or leg) function-related outcomes such as flexibility alongside potential neural (e.g. *H*-reflex activity) and structural (e.g. Young's modulus) modulators in the same study.

Secondly, varying experience with foam rolling may substantially modify the effects of the self-massage technique. Heiss et al. [57] demonstrated a stiffness decrease of the iliotibial band in experienced but not in novice foam roller users, which may indicate that both the local tissue and the central nervous system need to adapt to the loads applied. It is hence imperative to clearly describe the level of experience of the participants included into a study. Finally, all trials published hitherto included healthy active participants. On the one hand, assuming that tone and stiffness will be mostly normal in the majority of pain-free individuals, there may virtually have been nothing to release. On the other hand, in some cases, such release, if occurring, may even be harmful. The inclusion of healthy participants is also in sharp contrast to the claim of a myofascial "release" in active trigger points as it implies that the actual target population (patients with MTrP pain) has not been investigated so far. From a theoretical point of view, it is evident that if ROM was primarily inhibited by local MTrP, adhesions or spasms, rolling a distant, rolling an unaffected muscle should not have any effect on the mechanical properties of the targeted myofascia or muscles. Gathering data regarding this question will hence be an urgent task of future research in order to further substantiate the mechanistic foundation of rolling exercise.

7 Conclusions

Increases in ROM and decreases in pain sensitivity associated with foam rolling or roller massage have been attributed in many articles to a self-myofascial release of tissue stiffness, adhesions, scar tissue or spasms. The current evidence indicates that the term *self-myofascial release* is misleading and a misnomer. A misnomer such as self-myofascial release may become prevalent and ubiquitous since it was widely incorporated before the likely rolling mechanisms were determined and elucidated. Although there is some evidence suggesting local tissue-specific effects such as an increase in blood flow or a reduction in stiffness, these by far do not seem to represent the only mechanisms as the term self-myofascial release implies. Whereas manual forces are typically not sufficient to directly deform connective tissue (particularly if it is as strong as the iliotibial band), there may be a release occurring that is delayed via stimulation of proprioceptors or local hydration changes. This seems viable because all stiffness studies show a progressive decrease of stiffness but not immediately post-foam rolling, and this corresponds very similarly to the hydration dynamics [43]. A large body of research has unveiled that neuromodulatory responses seem to strongly affect the treatment response, particularly regarding the decrease of pain. The often observed global or non-local changes in pain provide strong evidence that mechanical alterations of a non-treated muscle or myofascia do not seem to be consistently present in healthy individuals and, thus, increased stretch tolerance effects on ROM due to activation of global pain modulatory responses such as DNIC and gate control theory as well as increased parasympathetic nervous system relaxation of muscle would be likely mechanisms. Thixotropic effects and decreased afferent excitability (*H*-reflexes) would contribute local mechanisms. In summary, at the present time, it needs to be concluded that more mechanistic research is needed to solidify the possible spectrum of mechanisms. Further research, preferably in patients and athletes with pain syndromes and or pathological alterations of soft tissue stiffness, should combine morphological and neural outcomes within the same study to clearly elucidate the mechanisms underlying rolling treatments.

Compliance with Ethical Standards

Conflict of interest Dr. Behm and Dr. Wilke declare that they have no competing interests.

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