



REVIEW: SYSTEMATIC LITERATURE REVIEW

Effects of self-myofascial release: A systematic review



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KEYWORDS

Foam rolling;
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Summary *Background:* Self-myofascial release (SMFR) is a type of myofascial release performed by the individual themselves rather than by a clinician, typically using a tool.

Objectives: To review the literature regarding studies exploring acute and chronic clinical effects of SMFR.

Methods: PubMed and Google Scholar databases were searched during February 2015 for studies containing words related to the topic of SMFR.

Results: Acutely, SMFR seems to increase flexibility and reduce muscle soreness but does not impede athletic performance. It may lead to improved arterial function, improved vascular endothelial function, and increased parasympathetic nervous system activity acutely, which could be useful in recovery. There is conflicting evidence whether SMFR can improve flexibility long-term.

Conclusion: SMFR appears to have a range of potentially valuable effects for both athletes and the general population, including increasing flexibility and enhancing recovery.

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Introduction

Myofascial release (MFR) has been described as an umbrella term for a wide variety of manual therapy techniques in which pressure is applied to muscle and fascia (McKenney et al., 2013). By extension, self-myofascial release (SMFR) is a type of MFR that is performed by the individual themselves rather than by a clinician, often using a tool. The most common tools used for SMFR are the foam roller (Kim

et al., 2014; Okamoto et al., 2014; MacDonald et al., 2013, 2014; Healey et al., 2014; Janot et al., 2013; Roylance et al., 2013; Peacock et al., 2014; Škarabot et al., 2015; Peacock et al., 2015) and the roller massager (Sullivan et al., 2013; Jay et al., 2014; Halperin et al., 2014; Bradbury-Squires et al., 2015). SMFR appears to have a wide range of effects. It is perhaps most well-known for increasing flexibility acutely (Mikesky et al., 2002; MacDonald et al., 2013; Sullivan et al., 2013; Roylance et al., 2013; Jay et al., 2014; Halperin et al., 2014; Bradbury-Squires et al., 2015; Peacock et al., 2014; Grieve et al., 2015; Škarabot et al., 2015) and chronically (Miller and Rockey, 2006; Mohr et al., 2014; Ebrahim and

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Elghany, 2013) by reference to changes in joint range of motion (ROM), although it has also been utilized to reduce delayed onset muscle soreness (DOMS) (MacDonald et al., 2014; Pearcey et al., 2014; Jay et al., 2014), affect arterial function and vascular endothelial function (Okamoto et al., 2014), and modulate autonomic nervous system activity (Kim et al., 2014; Chan et al., 2015).

Although SMFR appears to have various acute and chronic effects, there is currently no consensus regarding the exact mechanism or mechanisms by which SMFR leads to these effects, although many mechanisms have been suggested and reviewed in detail (Schleip, 2003; Simmonds et al., 2012). Most proposals regarding the potential mechanisms of action have focused on the nature of fascia itself (Schleip, 2003). However, exactly what is meant by fascia is difficult to specify because there are multiple definitions currently in use (Schleip et al., 2012; Langevin and Huijing, 2009), because fascial research is still in its infancy (Benjamin, 2009), and because the meaning of the word has changed over time (Langevin and Huijing, 2009). Fascia was recently defined in a review as 'fibrous collagenous tissues, which are part of a body wide tensional force transmission system' (Schleip et al., 2012). Indeed, the ability of fascia to transmit force has some support in the literature (Rijkkelijkhuizen et al., 2007; Meijer et al., 2007; Huijing and Jaspers, 2005; Stecco et al., 2008). Moreover, this definition may be helpful, as it differentiates fascia from connective tissue in general (Langevin and Huijing, 2009).

Despite difficulties with definitions, many important findings have been made regarding fascial tissues (Remvig et al., 2008) that provide clues to potential mechanisms by which SMFR might exert its effects. Fascia surrounds each muscle and organ in the body (Schleip, 2003); it is formed of numerous layers of collagen fiber bundles (Stecco et al., 2006); each layer contains parallel bundles while adjacent layers contain bundles at different orientations (Stecco et al., 2006); layers are separated by thin layers of adipose tissue (Stecco et al., 2006); and it is extremely strong (Findley et al., 2012) but plastic (Schleip, 2003). It has been reported that fascia displays piezoelectric effects (Yasuda, 1964), alters in stiffness following changes in water content (Chaitow, 2009), is richly innervated with nerve endings (Benjamin, 2009; Stecco et al., 2007), and contains many mechanoreceptors (Yahia et al., 1992). Fascia seems to be integrally involved in the biomechanics of the musculoskeletal system (Gerlach and Lierse, 1990), may be involved in force transmission (Benjamin, 2009), may contract like smooth muscle (Schleip et al., 2005), and can become inflamed and potentially thereby cause pain (Bednar et al., 1995).

In order to categorise the various potential mechanisms of massage, SMFR or MFR, reviewers have grouped fascia-specific mechanisms in different ways. Weerapong et al. (2005) categorized possible effects of massage into four types: biomechanical, physiological, neurological and psychological. Other reviewers have differentiated between two types: mechanical and neurophysiological (Schleip, 2003; Simmonds et al., 2012). Mechanical mechanisms of SMFR include thixotropy (Schleip, 2003), piezoelectricity (O'Connell, 2003; Schleip, 2003), fascial adhesions (Hedley, 2010; Martínez Rodríguez and Galáandel Río, 2013), cellular

responses (Chen and Ingber, 1999; Tozzi, 2012), fluid flow (Chaitow, 2009; Schleip and Müller, 2013), fascial inflammation (Bednar et al., 1995; Findley et al., 2012), and myofascial trigger points (Gerwin, 2010; Bron and Dommerholt, 2012). Many of these mechanical mechanisms have been criticized on the basis that pressures outside of normal human physiological ranges would be required in order to induce tissue deformations in most tissues (Chaudhry et al., 2008). Thixotropy is a process in which heat or pressure is applied to a material, which in turns makes it less dense and more fluid (Schleip, 2003). However, thixotropy is a transient and reversible effect (Mewis and Wagner, 2008). Consequently, it has been argued that thixotropy cannot explain the lasting changes that clinicians report from SMFR (Schleip, 2003). In the piezoelectric model, it is suggested that fibroblasts and fibroclasts, which create and digest the collagen fibers that are important for the biomechanical properties of the fascia, respond to electric charges created through pressure (O'Connell, 2003). While piezoelectric effects have been observed in collagen fibers for many years (Yasuda, 1964), it has been argued that it cannot explain the quick effects that clinicians observe (Schleip, 2003), which typically occur within 90–120 s (Barnes, 1997). In the fascial adhesions model, it is suggested that different fascial layers that would normally slide relative to each other alter such that they now stick to one another (Hedley, 2010; Martínez Rodríguez and Galáandel Río, 2013). These fascial adhesions are thought to be released by moving the body part through a full ROM under traction (Hedley, 2010). In the cellular responses model, it has been suggested that mechanical loading of fascia may lead to changes at the cellular level by reference to the principle of tensegrity (Chen and Ingber, 1999), in which it is proposed that cells are held in a state of continuous tension and respond to mechanical pressure by performing biochemical processes (Tozzi, 2012). In the fluid flow model, it has been suggested that since the water content of fascia affects its stiffness, and since fascia extrudes water when it is compressed, SMFR could increase the pliability of fascial tissues via temporary changes in water content that allow mobilization before the tissue rehydrates (Chaitow, 2009). The foam roller has been proposed as a tool particularly appropriate for this purpose (Schleip and Müller, 2013). Finally, models involving effects on fascial inflammation suggest that muscle or fascia may tighten as a result of inflammation (Bednar et al., 1995; Findley et al., 2012) and that SMFR might reduce this inflammation by increasing blood flow. Whether muscle or fascia can alter pathologically in this way is unclear but there are indications that SMFR and manual therapy in general can affect blood flow by increasing nitric oxide production (Queré et al., 2009; Okamoto et al., 2014). Such fascial inflammation may be related to the concept of myofascial trigger points, which have been proposed to occur when motor endplates release excessive acetylcholine, shortening sarcomeres locally, disrupting cell membranes, damaging the sarcoplasmic reticulum, and causing inflammation (Hong and Simons, 1998; Gerwin, 2010; Bron and Dommerholt, 2012). However, the phenomenon of myofascial trigger points has been drawn into question by concerns over the reliability of their clinical identification (Myburgh et al., 2008).

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