Myofascial pain definitions, historical perspective and further developments

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This article offers a brief overview of myofascial trigger point (MTrP) definitions and informs the clinician of an often unknown aspect of the historical basis for myofascial pain syndrome (MPS) and MTrP theory. It is hoped that the further developments section will encourage the reader to explore some of the later advancements and possible gaps in the evidence in relation to MPS and MTrPs, and increase their critical understanding of this approach. I have purposely not focused the content on the clinical efficacy, or related evidence in respect of myofascial pain / MTrP therapy, in order to enable a more comprehensive approach on an important aspect of myofascial pain theory. However, the article does present some of the important background and further development evidence that may enable and critically inform your clinical practice in relation to myofascial pain and MTrP therapy, and in line with the goals of championing evidence based cost effective physiotherapy.

Learning outcomes

1. Encourage the exploration of advancement and possible gaps in the evidence.
2. Increase critical understanding.
3. Understand the important aspect of the theory of the subject.

Definitions

The most commonly used definition for myofascial trigger points (MTrPs) is that by Travell & Simons (Travell & Simons 1983; Simons *et al* 1999), who define them as hyperirritable areas within taut bands of skeletal muscle or fascia. MTrPs are further classified as active or latent in nature. An active MTrP produces symptoms that can trigger local or referred pain, whereas a latent MTrP does not trigger pain unless being stimulated (Dommerholt *et al* 2011). Active MTrPs found in a specific region of the body such as the neck, back and shoulder, are often referred to as a myofascial pain syndrome, or MPS (Simons *et al* 1999).

MTrPs feature the hallmark characteristics of MPS and may exhibit motor, sensory and autonomic components (Dommerholt *et al* 2011). The motor component of active and latent MTrPs may include restricted range of motion (ROM) (Grieve *et al* 2011; Grieve *et al* 2013). Latent MTrPs may also alter the timing and decrease the consistency of the muscle activation pattern (MAP) of scapular rotator muscles (Lucas *et al* 2004, 2010), and may inhibit normal motor activity in the muscle of origin or in functionally related muscles. Motor inhibition is often identified clinically as muscle weakness, poor co-ordination and muscle imbalance (McPartland & Simons 2011); although MTrP motor effects have gone largely unrecognised because of a lack of published research studies (McPartland & Simons 2011).

MTrPs may also be associated with autonomic signs including changes in skin temperature, skin colour, piloerection (goosebumps) and lacrimation (shedding of tears) (Gerwin 2010). Sensory components may include local tenderness, referred pain, and peripheral and central sensitisation (Dommerholt *et al* 2011). Peripheral sensitisation is a reduction in threshold and an increase in responsiveness of the peripheral nocioceptors, central sensitisation is an increased responsiveness of neurons in the central nervous system (CNS) (Dommerholt *et al* 2011). The signs of peripheral and central sensitisation are hyperalgesia (an increased response to a stimulus that is normally painful), allodynia (pain due to stimulus that does not normally provoke pain) and referred pain, which may become chronic and widespread (Meeus & Nijs 2007; Dommerholt *et al* 2011).

Further research is needed to investigate the relationship between central sensitisation and MTrP formation as there is no evidence to support that central sensitisation induces the development of new

MTrPs (Ge *et al* 2011). Recent exploration by Fernández-de-las-Peñas and Dommerholt (2014) into whether TrPs are a peripheral or central phenomenon highlighted, in summary, the growing literature supporting the concept of TrPs starting as a peripheral process that can contribute to and maintain central sensitisation.

In relation to MTrP definitions and terminology is the growing awareness of fascia and its role in musculoskeletal dysfunction. Fascia is a connective tissue which surrounds every nerve, blood vessel and muscle fibre in the human body resulting in the connection of bones, muscles and organs which form large networks throughout the body (Schleip *et al* 2012a). Based on the tensegrity principle, previous studies have highlighted the presence of continuity and connectivity between fascia or muscle that may be anatomically distant from each other (Langevin 2006; Kassolik *et al* 2009).

The tensegrity principle is a proposed concept of muscular skeletal relationships which refers to the forces of tension provided by muscles, tendons, ligaments and fascia on bones and joints. It is not within the scope of this article to fully develop the complex concept of tensegrity. Fuller explanations are provided by the following influential fascial texts: *Anatomy Trains* (Myers 2014) and *Fascia: The Tensional Network of the Human Body* (Schleip *et al* 2012b).

Anatomical dissections have confirmed the continuity of the fascial system in the upper and lower limbs (Stecco *et al* 2007, 2008). Anatomy trains, a schematic map of the body's fascia connections, has suggested and proposed that any tension at a particular part of an "anatomy train" may have detrimental effects resulting in global decreased flexibility (Myers 2014). Myofascial therapies cover a numerous and varied spectrum of techniques, including osteopathic soft-tissue techniques, structural integration (Rolfing), massage including connective tissue massage (CTM), instrument assisted fascial release, MTrP therapy, strain / counter-strain and muscle energy technique (MET) (Simmonds *et al* 2012).

In clinical practice, it is sometimes difficult to ascertain if a fascial or MTrP intervention will target only the fascia, or reach both the underlying muscle and associated MTrPs. Specific treatments aimed at the fascia may need to review the possible impact on MTrPs and be aware of the integral link between muscle and enveloping fascia (epimysium), muscle fibre bundles (perimysium), and individual muscle fibres (endomysium) (Dommerholt 2012).

The early historical perspective

The evolution of the term MTrP can be linked to the unknown cause of muscle pain developing in an otherwise fit person which, for centuries, remained an enigma and consequently meant that no general agreement on what to call the underlying disorder could be reached (Cummings & Baldry 2007). Through the years, individual authors have taken a cluster of signs and symptoms and allocated them a variety of names or, even more confusingly, assigned different signs and symptoms to something with an already established name (Simons 1990), for example:

- muscle callus (Froriep 1843)
- muscular rheumatism (Adler 1900)
- fibrositis (Gowers 1904; Kelly 1941)
- myalgic spots / nodules (Good 1942)
- spot tenderness (first description of MTrPs by Travell *et al* 1942).

Some of these listed historical terms and authors have been identified and cited by Simons *et al* (1999) as evidence for the evolution of MTrP theory.

Following an experimental study on referred pain from muscle (Kellgren 1938), three clinicians, researching independently on three different continents (Kelly 1941; Good 1942; Travell *et al* 1942), published a series of studies emphasising four cardinal features of a MTrP:

- a palpable nodule or band like hardness in the muscle
- a highly localised spot of extreme tenderness in that band
- reproduction of the patient's distant pain complaint by digital pressure on that spot
- relief of the pain by massage or injection of the tender spot.

According to Mense and Simons (2001), these three publications (Kelly 1941; Good 1942; Travell *et al* 1942) were pivotal in the development and background to our understanding of MPS caused by MTrPs. In reviewing each of these three proposed pivotal studies in the evolution of the MTrP concept (Kelly 1941; Good 1942; Travell *et al* 1942), it is clear that the authors paid tribute to the influential work of Kellgren (1938). All three had identified MTrPs. However, each used different diagnostic terms to identify and describe them.

A preliminary observational study on referred pain arising from muscles of normal subjects was performed by Kellgren (1938). These experiments were conducted in the laboratory of Sir Thomas Lewis (University College London) and involved Kellgren injecting a hypertonic saline solution into specific muscles on himself and other workers in his laboratory. Kellgren (1938) based his study on previous work by Lewis (1938), who had found that injecting substances (not specified) into muscle had the identical effect to that resulting from ischaemic conditions and, therefore, was claimed to represent true muscle pain.

To ensure muscular pain was being assessed, Kellgren (1938) used Novocaine to anaesthetise the overlying skin before injecting a saline solution into the muscle. The investigation included muscles of the trunk, hand, lower and upper limb, and concluded that fascia and tendon sheaths give sharply localised pain, while muscle gives diffuse pain which may be referred (Kellgren 1938). Any underlying causes of the referred pain, such as overuse, trauma, or surgery were not made clear, but further findings were that diffuse pain from a given muscle is always distributed within certain regions, although the distribution within these limits varies between individuals (Kellgren 1938). The distribution of the diffuse muscle pain appears to follow a spinal segmental pattern and that pain from muscle may be confused with pain arising from deep structures such as the joints (Kellgren 1938).

Kellgren (1938) concluded that the mechanism of pain referral from muscle and joints follows a final common path in the CNS. This early view of the possible mechanism of muscle pain and nature of MTrPs was echoed years later by Lucas *et al* (2004) who stated that MTrPs are not just contracted muscle fibres, but neuromuscular lesions that form part of the neurological loop that affects, and is affected, by the CNS.

All experiments by Kellgren (1938) were performed on asymptomatic individuals and pain was induced by the introduction of saline solution. This influential study in the field of myofascial and MTrP pain illustrates the possible confusion between the origin of pain (differential diagnosis joint and muscle) and the fact that pain can be referred from muscle. Whether injecting localised saline can be compared to MTrP, e.g. nodule, taut band, localised tender area, in its signs and symptoms is an important question. The research by Kellgren (1938) provides a foundation for the understanding of the existence of MTrPs.

Kelly (1941) investigated and treated 200 cases of somatic pain (fibrositis) that were with, or without, co-existing pain in the region of joints. The main assertion in the case series was the presence of a muscular lesion (nodule) in all cases of pain of uncertain origin. Nodules were found in the:

- gluteus maximus, erector spinae (lower back pain)
- gluteus maximus near ischael tuberosity (sciatica)
- intercostals, erector spinae (chest pain)
- upper trapezius, occipital region (neck pain)
- anterior deltoid (upper extremity pain)
- wrist extensors (tennis elbow)
- medial longitudinal arch muscles (foot strain)
- soleus, gastrocnemius (knee joint pain).

Some of these observations on muscular nodules correlate with more recent findings by Travell & Simons (1983; 1992) and Simons *et al* (1999) in which mapping of MTrPs and regional pain was undertaken.

Good (1942) identified myalgic spots and pain referral patterns for 20 muscles in the upper and lower quadrant, in 500 cases of myalgia in the British army. The objective signs for myalgia revolved around myalgic spots, which were well-defined tender areas in the origin, insertion and course of muscle. Good (1942) linked these myalgic spots with well-defined nodules that may, or may not, be present in the muscle. Pressure on these myalgic spots elicited a severe pain often similar to the spontaneous pain complaint and a pathogenic sign of wincing, or involuntary movement similar to the "jump sign", which was identified as a diagnostic criterion of MTrPs in later work by Travell & Simons (1983) and Simons *et al* (1999). In findings similar to those of Kellgren (1938), Good (1942) concluded that referred pain was often dermatomal in distribution, and from myalgic spots in muscles supplied by the same spinal segments.

In a further case series, Travell *et al* (1942) recruited 58 people with shoulder and / or arm pain. In the majority of cases the onset was gradual, with precipitating factors ranging from physical fatigue, chilling, infection, trauma, muscular inactivity and poor posture (protracted shoulder girdle). In stark contrast to Kelly (1941) and Good (1942) who referred exclusively to nodules and myalgic spots, Travell *et al* (1942) used the term trigger point (TrP) and described how all the cases presented with one or more TrP. The main criteria for identification of a TrP included excruciating tenderness on strong pressure, pressure eliciting pain in the reference zone, active contraction or passive stretching of the muscle inducing referred pain. Apart from serratus posterior and infraspinatus, for which referral patterns were clearly linked to their dermatome distribution, Travell *et al* (1942) asserted that reference patterns did not clearly align with somatic reference zones mapped out for single spinal segments (Kellgren 1938), and concluded that idiopathic myalgia best described the syndrome of a restriction of motion primarily as a reaction to pain. This is the first mention of the word syndrome that may provide evidence for the existence of MTrPs and related muscle pain and, as previously asserted, one of the key definitions in MTrP literature is the related term MPS.

Dr Janet Travell, the author of this pivotal study in the history and understanding of MTrPs, was a cardiologist treating patients with life-threatening pulmonary disease (Mense & Simons 2001). Some of her patients complained more about the devastating pain in their shoulders and arms than about their major illness and, on examination through systematic palpation of the scapula and chest muscles, Travell uncovered the presence of trigger areas. In her autobiography, *Office Hours: Day and Night* (1968), Travell noted that palpation of her own shoulder pain and presence of "sore spots" was her first introduction to "enigmatic trigger areas". It was at this time that she read a study by Edeiken and Wolferth (1936) on persistent pain in the shoulder following myocardial infarction and, like previous clinicians (Kelly 1941; Good 1942) she was influenced by the findings of Kellgren (1938).

Further to the three previously mentioned influential case series, Travell and Rinzler (1952) published a study on "The Myofascial Genesis of Pain", which reported pain patterns of MTrPs in 32 skeletal muscles from a sample population of 1,000 patients with MPS. As in Travell *et al* (1942), there was minimal explanation of the histology and pathophysiology, and nothing relating to the exact mechanism of pain referral. The 32 muscles and TrP referral patterns were later included in the two volume book entitled *Myofascial Pain and Dysfunction, The Trigger Point Manual* (Travell & Simons 1983; Travell & Simons 1992) and again in the later second edition (Simons *et al* 1999).

The historical literature is largely based on a multiple case study, or case series approach, the design and method of which may help in the discovery of new diseases, provide new ideas in medicine (Vandenbroucke 2001), have the advantages of replication across a number of cases, and have the ability to strengthen theory (DePoy & Gitlin 2005). However, there are limitations of a case series in respect of the descriptive nature, lack of a control / comparison group and susceptibility to bias (Kooistra *et al* 2009).

Although the publications mentioned stem from the 1930s and 40s, it could be argued that the basis of MTrP theory has not

changed considerably to the present day. The early pioneering work, specifically by Travell and later in collaboration with David Simons, has remained the foundation of MTrP theory.

Further developments

While the focus of this article is primarily on an aspect of the historical basis of MPS and MTrP theory, the reader is advised to explore some of the later developments, related key evidence to support your use and / or critical appraisal of MTrP therapy, and possible gaps in the literature in relation to the following:

- pathophysiology of MTrPs integrated hypothesis (Simons *et al* 1999)
- presence of noxious chemicals in MTrPs as a possible explanation for the persistence of pain associated with MTrPs (Shah *et al* 2005, 2008)
- absence of laboratory methods or imaging to objectively confirm or diagnose a MTrP, developments in Magnetic Resonance Elastography (MRE) (Chen *et al* 2007); Doppler Ultrasound (Sikdar*et al* 2009) and advancements in ultrasound elastography (measures tissue stiffness) in relation to MTrP diagnosis (Brandenburg *et al* 2014)
- reliability issues in relation to physical examination and palpation for the diagnosis of MTrPs (Lucas *et al* 2009; Myburgh *et al* 2008)
- increased discussion in relation to peripheral and central sensitisation as an explanation for the chronicity and referred pain in MPS (Ge *et al* 2011; Fernández-de-las-Peñas & Dommerholt 2014).

The later developments and related key evidence described previously are an example of the increased research evidence in today's MPS / MTrP literature. The current research evidence uses many types of design, from case studies to multi-centre randomised controlled trials (RCT). Some of the available evidence is of high quality and some has limitations in respect of scientific rigour and associated internal and external validity issues.

Importantly, the focus of this article is not on the clinical efficacy, or related research evidence in respect of myofascial pain and MTrP therapy for specific conditions or regional pain. Instead, the aim is to provide you with some of the important evidence based definitions, historical background, later developments and related key research evidence that may critically inform your clinical practice in relation to myofascial pain and MTrP therapy. A recent evidence-informed review of the current myofascial pain literature included publications originating from many countries, including Australia, Brazil, Columbia, Denmark, Italy, Iran, Israel, Japan, New Zealand, Qatar, South Korea, Spain, Turkey, the UK and the United States, which reflects the widespread interest in myofascial pain and related research evidence (Dommerholt *et al* 2015).

About the author

Rob qualified as a physiotherapist after seven years as a podiatrist in the NHS. His physiotherapy experience has, since, included working with Bath Rugby Academy, the NHS and private work. He has been involved as an examiner, and is currently a visiting lecturer on the Bath University MSc Sports Physiotherapy programme.

He completed a PhD on myofascial trigger points (MTrPs) in the triceps surae and is actively involved in myofascial pain, fascia and musculoskeletal research and clinical application of MTrP therapy. Previous MTrP publications have been in the journals of *Bodywork and Movement Therapies, Physiotherapy* and *Manual Therapy*. He has presented at national and international conferences and is a conference and journal peer reviewer. Currently, Rob is part of a team of authors involved in the quarterly published "an evidence-informed review of the current myofascial pain literature" within the *Journal of Bodywork and Movement Therapies*. Rob has experience in running MTrP courses throughout the UK and Ireland.

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