# A Better Way to Ease the Pain for The Lower Back: Dynamic Myofascial Release

A Research assignment presented to
The Faculty and Students studying Diploma of Remedial Massage
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By
Hill Yang
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# Abstract

Muscular pain and injury occur when the musculotendinous is exposed to single or recurrent episodes of biomechanical overloading. Injured muscles are usually abnormally shortened with increased tone and tension due to varied states of over contraction. Injured muscle often meets diagnostic criteria for a “myofascial pain syndrome”.

There’re many ways to deal with the muscle shortening, the majority way is stretching, including 7 types of stretching which are, static stretching, passive stretching, dynamic stretching, ballistic stretching, active isolated stretching, isometric stretching, and proprioceptive neuromuscular facilitation(PNF). All of them want to reach our maxima ROM, too much ROM will decrease our performance and joints stability[29-31] , irrespective of the stretching technique used[32,33]. For people who got normal ROM doesn't mean they can feel pain-free or can master any kinds of exercise. When we learn a new skill, the motor, tissue and physiological adaptation is specific for that task(specificity),[35-37]. This allows the task to be performed optimally with minimal energy expenditure physical stress and error. This adaptation is profoundly unique, optimized for that particular activity but often unsuitable for a different activity[38,39-41].

It is this dehydration of the tissue,with the accompanying development of cross-links at the nodal points, that can put enormous and excessive pressure on pain- sensitive structures and limit the fascial system’s ability to glide. This enormous pressure, approximately 2,000 pounds per square inch, can produce symptoms of pain, including headaches, fibromyalgia, and limited motion[73].

Everyone is different so their Functional Range of Movement(FROM) are different as well, so my goal is to help patients to reach their FROM. Another part of stretching, the force to failure was 15% higher and the energy absorbed was 100% higher in muscles stretched to failure while activated[27]. That’s one of reason that PNF and MET is so effective to achieve maximum flexibility.

To ease the muscular and the myofascial pain syndrome, myofascial release is the one of the best way to reach the goal, so I design a technique to work on FROM and MFR in the same time to activate the muscle and release the fascial tension, and I called it Dynamic Myofascial Release(DMFR).

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# Background

## Define the Pain

Muscular pain and injury occur when the musculotendinous contractual unit is exposed to single or recurrent episodes of biomechanical overloading. Injured muscles are usually abnormally shortened with increased tone and tension due to varied states of over contraction. Injured muscle often meets diagnostic criteria for a “myofascial pain syndrome”, a condition originally described by Drs Janet Travell and David Simons.[1] Myofascial pain is estimated to account for 85% of muscular pain due to injury[2] and 90%[3] of patients treated in pain clinics. A Danish study found that myofascial pain was present in 37% of men and 65% of women within a randomly selected population of 1504 people aged 30-60 years.[4] Over 44 million Americans are estimated to have this condition at a coast of $US47 billion per year.[5.6] Despite these impressive statistics, much disagreement exists between physicians and other medical specialists regarding the diagnostic criteria for myofascial pain and its very existence as a pathological entity.[7] Current theories regarding the cause of myofascial pain will be presented, followed by a review of clinical management strategies.

## 1.Muscle Pain

Pain receptors in muscle are sensitive to a variety of mechanical stimuli including pressure, pinching, cutting or stretching.[8] Acute muscular pain, with duration of >6 weeks, can result from single episodes of macro trauma or recurrent microtrauma, especially when biomechanical overloading of the muscle occurs during the active contraction or stretch phase.[8,9] Muscular contusions and lacerations are typically caused by macro trauma, whereas delayed onset muscular soreness and strain usually result from repetitive overloading.[9] Delayed-onset muscle soreness is defined as muscular pain that occurs 24 to 72 hours after vigorous exercise. It is common following unaccustomed intense exercise and is characterized by discomfort beginning several hours after exercise and peaking after 1 to 3 days. This entity can be distinguished from acute muscle strain, which is characterized by immediate pain associated with diminished function. Delayed-onset muscle sore- ness will resolve in a few days and requires no specific treatment.[9] Musculoskeletal pain is designated as chronic after 12 weeks.[8,10,11] Chronic muscular injury may be caused by repetitive activities that cause cumulative overwork of specific musculotendinous groups. Muscles that brace or support working muscles are at risk for painful strain, as well as muscles that have reciprocal antagonist-agonist relationships within the kinetic chain.

Generalized musculotendinous pain can occur when biomechanical loads are shifted from painful structures to neighboring joints and muscles within the kinetic chain secondary to compensatory overuse, i.e. low back pain following an injury to the hip or knee.[8] Also, pain spread may result when agonist muscles are overexerted to compensate for a lack of assistance usually received by another muscle group that shares a specific movement, i.e. mouth closure augmented by ipsilateral temporals muscles to rest an inefficient painful master muscle in spasm. Fibromyalgia is a frequently diagnosed disorder characterized by diffuse somatic hyperalgesia and diagnostic guidelines have been established by the American College of Rheumatology.[12] Unlike myofascial pain, fibromyalgia affects both non contractile and contractile connective tissues, and tender muscles do not necessarily contain trigger points. Recent research has revealed

additional biomechanical and neurophysiological influences that facilitate the spread of pain to other tissues and body areas.[13-16]

Nociception is the neurochemical process whereby acute tissue damage activates specific nociceptors which convey pain signals through peripheral neural pathways to the CNS. Transduction is the process whereby noxious afferent stimuli are converted from chemical to electrical messages within the spinal cord that communicate cephalic to the brainstem, thalamus and cerebral cortex.[17] Peripheral and central sensitization occurs through release of substance P and activation of NMDA receptors.[18] Chronic nociception may recruit sympathetically mediated activity which augments pain intensity and causes autonomic dysfunction, such as abnormal sweating or vasomotor instability.[10] Therefore, the intensity and character of pain that is initially generated from a peripheral pain generator, such as muscle, can be influenced by multiple neurochemical mechanisms, which in turn cause more durable and enduring nociceptive activity.[8,16]

# 2.Myofascial Pain

## 2.1 Definition

Myofascial pain is characterized by muscles that are in a shortened or contracted state with increased tone and stiffness, and that contain trigger points.[1,8] Trigger points are tender, firm nodules 3-6mm in size that are found on placatory examination.[1] Trigger point palpation provokes radiating, aching type pain into localized reference zones. Mechanical stimulation of the “taut band”, a hyper irritable spot within the trigger point, by needling or rapid transverse pressure will often elicit a localized muscle twitch. Sometimes trigger point palpation can elicit a “jump sign”, an involuntary reflex or flinching of the patient that is disproportionate to the placatory pressure applied.[1,8] The natural history of active and asymptomatic trigger points is unknown.[1] Myofascial pain and dysfunction may become symptomatic from direct or indirect trauma, exposure to cumulative and repetitive strain, postural dysfunction, and physical deconditioning.[8]

## 2.2 Pathogenesis

The pathogenesis of myofascial pain and trigger points remains unproven. Hubbard and Berkoff found increased spontaneous activity and spike disharges in trigger points when examined by needle electromyography(EMG). They postulated that this electrical activity, discovered by EMG, was generated by efferent sympathetically induced hyperactivity of the muscle spindle.[19,20] Hbbard theorized that EMG recorded spikes in the trigger point are recorded from intrafusal muscle fibers,[20] as opposed to the assumption by most electromyographers that these spikes occur in the sarcolemmal endplate as a result of the firing of single extrafusal muscle fibers from needle irritation.[21,22] Pharmacological s tudies reviewed by Hubbard revealed that ∂-adrenoceptor antagonist phentolamine temporarily reduced trigger point electrical activity when injected intravenously or intramuscularly  in the vicinity of the trigger point.[20] Trigger point injections with another ∂-adrenoceptor antagonist, phenoxybenzamine, demonstrated longer duration of pain reduction that lasted up to 4 months after injection.[20] Although these studies seemed promising, some study participants developed subcutaneous fat necrosis at the injection sites. Furthermore, to date, double-blind controlled research that supports the use of phenoxybenzamine for myofascial pain treatment has not been published.

A recent review of the neurophysiology of myofascial pain cited anecdotal EMG findings that support the idea that trigger points may represent an are of focal dystonia.[21] Animal studies show that trigger points can be abolished by transaction of efferent motor nerves or infusion of lidocaine; however, spinal transaction above the level of segmental innervation of a trigger point-containing muscle does not alter the trigger point response.[21] To date, research suggests that myofascial dysfunction with characteristic trigger points is a spinal segmental reflex disorder.[21]

Simons postulates that abnormally increased production and excessive release of acetylcholine at the neuromuscular junction causes sustained depolarization of the post-junctional muscle cell membrane under resting conditions with persistent shortening of sarcomeres.[1] Resultant muscle spasm may impair arterial inflow, and with it the supply of oxygen, calcium and other nutrients necessary to induce energy-dependent muscle relaxation and to meet the higher energy demands required by aberrantly sustained muscle contraction.[1] In addition, continued contractile unit shortening and spasm can cause distortion and damage of involved tissues, which may precipitate the synthesis and release of endogenous allogenic biochemical and inflammatory substances that enhance nociception.[23-25]

## Ease The Pain

Most treatment methods for myofascial pain are empirical and aimed at the painful trigger point with the purpose of ablating muscle spasm and restoring normal muscle length, function and strength.[26]

## Restoration of muscle strength

After the resolution of the acute pain and swelling, most authors recommend the institution of a program of physical therapy. This is beneficial for restoring normal muscle strength and flexibility to the muscle.

Restoration of muscle strength is important to prevent further injury or reinjury because of the role of muscle as an energy-absorbing structure. The ability of the muscle to resist lengthening is a measure of its capacity for energy absorption. Muscle can do this in two ways: passively, by the resistance of the connective tissue elements within the muscle, and actively, by contraction against the lengthening force.

These concepts have been demonstrated in a laboratory study of rabbit muscles stretched to failure in the activated and nonactivated states.[27] In that study, the force to failure was 15% higher and the energy absorbed was 100% higher in muscles stretched to failure while activated. At small deformations of the muscle, most of the energy absorption was due to the active component rather than the passive component. Most physio- logic activity in eccentrically contracting muscle occurs at relatively small deformations. Thus, muscle weakness should significantly impair the ability of the muscle to absorb energy, making it more susceptible to muscle strain injury. Continuing this logic, returning an injured muscle to full strength is important in preventing additional injury.

Passive stretching of muscle is thought to be beneficial because it reduces muscle stiffness. A laboratory study showed that much of the decreased stiffness is due to viscoelastic properties rather than reflex changes.[28] Because of viscoelasticity, prolonged stretching can lead to diminished stress within the muscle for a given length change. Although the temporal characteristics of this effect are unclear, this property

represents a plausible mechanism by which stretching might prevent further muscle strain injury in the postinjury setting.[9]

# Stretching

## Stretching and human movement performance

Another common belief is that human performance can be enhanced by an acute bout of warm-up stretching before a competition or, in the long-term, by regular stretch training.

If warm-up stretching were beneficial for enhancing performance we would have expected nature to have “factored-in” stretching as part of animal behavior. However, with the exception of humans, no other animal seems to perform pre-exertion activities that resemble a stretch before they chase their prey, and, reciprocally, the prey dose not halt the chase for the lack of a stretch. The stretch warm-up in humans seems to be largely ceremonial. A person would stretch in the park before a jog but would not consider stretching to be important before sprinting for a bus. A person my stretch before lifting weights in the gym but a builder is unlikely to stretch although they may be lifting and carrying through the day. We have evolved to perform maximally, instantly and without the need to limber up with stretching. It does not seem to confer any biological advantage nor is it physiologically essential to performance.

This observation is reflected in research that looks at stretching in sports. Stretching as a warm-up before and after alleviating muscle soreness, it provides no protection against sports injuries and vigorous stretching before an event may even reduce sports performance[29-31]. It was demonstrated that strength performance can be reduced by between 4.5% to 28%, irrespective of the stretching technique used[32,33].

It seems that animals do what they do without having to use training that is outside their movement repertoire[34]. They develop their functional movement repertoire by simply doing their species-specific activities and nothing more. Birds learn to fly by flying and optimism flying by more flying. This behavior is called task-specific practice or functional training [34]. They don’t do wing press-ups or stretching, termed here extra-functional training.

If regular stretching could improve a wide range of unrelated activities it would mean that a very agile individual, such as a ballerina, would excel in all sports activities. But how likely is a ballerina to exhibit high levels of performance in running, swimming, cycling, football or weight training, swimming, cycling, football or weight training, activities for which stretching is often recommended? Why don’t we see ballerinas and yoga instructors? winning medals in the Olympics? To answer these question we need to look at an adaptation phenomenon called specificity.

## Specificity in adaptation

When we learn a new skill, the motor, tissue and physiological adaptation is specific for that task(specificity),[35-37]. This allows the task to be performed optimally with minimal energy expenditure physical stress and error. This adaptation is profoundly unique, optimized for that particular activity but often unsuitable for a different activity[38, 39-41]. This means that training gains are task-specific. They do not seem to carry over or transfer to an activity which is dissimilar. Even activities that look identical, such as sprinting and distance-running each have their unique, biomechanical and physiological non-transferable  adaptation. The knee force angle profile is different between these activities, and the leg muscles in springers have greater fascicle length and lesser penna-

tion angle than in distance runners [42]. Furthermore, studies in motor control over the period of a century have also demonstrated that such task specific adaption takes place in the central nervous system including the spinal cord and brain centers [43]. So, sports specific adaptation is a whole body phenomenon that includes peripheral musculoskeletal and central control changes. that is why sprinters don't make great marathon runners and vice-versa.

Specificity and transfer has been studied extensively in sports with the aim of identifying training methods that could improve sport performance. Overall, most studies show a lack of transfer and, on the occasion when it is demonstrated, it seems to be of marginal effect and unpredictable. For example, sprinting performance was shown to improved by training in vertical but not by training in sideways jumps[44]. Transfer may fail to occur even in training systems that closely resemble the task. For example, resistance sprint training using a towing device fails to improve sprint performance. Similarly, off-ice skating exercises do not improve on-ice performance in speed skaters. Resistance training in swimming results in a different swimming results in a different swimming style using large amplitude trunk movement that may not be beneficial for free, unrestricted swimming [45]. Core stability exercises fail to improve sports performance[46] [47,48]. Different forms of resistance exercise have failed to improve sports-specific activities such as football kicks[49], throwing velocity in water polo[50] and rowing [51]. Even resistance training in one particular posture may not transfer force gains to other postures[52]. Cross training by cycling does not improve running and may even reduce running economy[53,54]. Training in isolated tasks, such as hip flexibility or trunk strengthening activities, does not improve the economy of walking or running [55]. Upper limb resistance exercises do not improve arm coordination[56], and so on.

From a specificity perspective, regular stretching is unlikely to improve the performance of a dissimilar task. Indeed, to date, the evidence suggests that regular stretch training does not improve sports performance and may even be reduced by acute bouts of stretching[57][58]. The sport specific adaptation is likely to be very different from the musculoskeletal adaptation induced by stretch training. It is, therefor, unlikely to meet the physiological demands of the particular sport activity. At best stretching may have no effect but at worst, it may introduce a "competition in adaptation" if the individual is excessively focusing on their extra-functional stretching rather than their sports specific training[34,43]. Under these circumstances stretching is very likely to have a negative effect on performance.[59]

## Defining functional and dysfunctional ROM

A patient who presented to my clinic complained of several months of moderate shoulder discomfort during the night. On examination, there was total loss of external rotation on the affected side, but all other ROMs were normal. Interestingly, the patient was unaware that she had such profound ROM losses, in particular as she was still able to carry out all her daily activities to the full. She was only concerned about the pain that kept her awake at night. This case suggests that ROM can be evaluated by using a clinical-anatomical and functional reference points. A clinical-anatomical model often uses the unaffected side or published ROM values as a reference for comparison. [60-65] A functional assessment explores how ROM changes impact the patient's ability to perform a range of daily activities. These two forms of evaluation are not necessarily linked and But what do we mean by functional ROM?

## Functional ROM

Functional movement is the unique movement repertoire of an individual [66] This repertoire contains some shared movement patterns associated with daily needs and demands such as feeding, grooming and travelling. Some of the functional repertoire is particular to the individual, containing specialized occupational and recreational activities or sport pursuits. Within functional movement is the person's functional ROM. It is the ROM required to perform functional activities effectively, efficiently and comfortably; [67] to have sufficient ROM to reach for a shelf, bend to lift or hip flexibility to walk (shared activities), or the extra flexibility needed for a dancer to perform full splits (special activities). Most individuals use a relatively small percentage of their full active ROM when performing shared activities[68-73] Hence, these are often performed within a comfort zone, with relative ease and without any stretch discomfort or pain. Infrequently, some shared activities may challenge the margins of this comfort zone, such as the full lumbar flexion required to pick an object off the floor or the full cervical rotation needed for reversing the car.[68, 69, 73] Special activities, such as yoga or dance, require greater functional ROM and are often expected to be performed with some level of discomfort and effort. How far a person chooses to move into ranges that are uncomfortable and even painful depends on their movement goals and pain tolerance. These individuals may consider movement in this extended zone to be normal and even desirable.

## Dysfunctional ROM

Often a person will become aware of ROM losses when they are no longer able to perform functional activities to the full. Hence, the use of functionality as a reference point can also help to define dysfunctional ROM: ROM limitation that impedes the ability to perform functional movement . The value an individual gives to any ROM loss is closely related to how adversely it affects their functionality. ROM losses that affect shared activities are obvious, noticeable and likely to be considered dysfunctional by the person concerned. An example would be loss of hip extension affecting walking a bility. However, a small loss of full shoulder flexion may go undetected unless the person is attempting to perform a special activity, such as painting a ceiling or doing a hand stand. Furthermore, a minor ROM loss for one individual may be experienced as a serious impediment for another. The loss of full shoulder flexion would be more limiting and distressing for a dancer than, say, a footballer. So whether ROM is functional or dysfunctional is determined by the individual's expectations and the requirement of their functional repertoire. This brings us back to the original question in the introduction; is the lac k of flexion ROM in forward bending functional or dysfunctional? That depends on what t he person is trying to achieve. There is no ROM "pathology" as long as the stiffer individual is comfortable and able to perform daily activities that require some degree of for ward flexion, such as putting on their socks. They are still likely to be within their functional ROM even if they take up a new sport that does not require any special forward be nding flexibility, such as running. If they took up yoga their limited flexion ROM would be come an impediment, but only in relation to this new activity. Their other shared daily activities would remain unaffected and therefore functional.

## Functional and clinical ROM ideals

The use of functionality as a reference point can also help to define treatment suc cess and to some extent determine the treatment ending. Most patients commence their therapeutic journey in the hope of regaining their health; ideally, as they were before the

onset of their condition. Functionality is often their reference point for evaluating improvements and treatment success. This may even be in the absence of full ROM recovery. For example, a patient with frozen shoulder may recover full functionality, but on examination the therapist observes a residual 15–20 ° loss of flexion. The patient considers the treatment a success but the therapist with the medical knowledge does not. It could be argued that, since this residual loss does not impede functionality, recovering it remains a clinical ideal but an irrelevance to the patient. For example, it was found that a fter knee replacement the majority of patients obtained a flexion greater than 115 ° while some obtained flexion greater than 125 °.[74] However, there was no difference between the two groups on functionality scores. So what do we do, terminate the treatment once functionality is attained or continue until ROM is fully recovered? Ultimately, t he decision depends on the patient's expectations and how the loss limits their functional repertoire. But it also depends on their knowledge of their condition and their beliefs a bout flexibility. Many patients (and therapists) believe that residual losses will result in so me joint pathology and disability later in life. Because of this fear some will pursue the cli nical ROM ideal and choose to continue the treatment. These fears can be alleviated by reassuring the patient that such longterm consequences are unlikely and that, in time, n ormal daily use will promote recovery. So, often a well-
informed patient would choose to end the treatment when the ROM has sufficiently recovered to perform functional tasks. However, some patients' expectations are set within the clinical ideals. They would like to have full ROM. All this does not exclude the possibility that in some situations the clinical ROM ideals are essential treatment goals, i.e. f ull ROM recovery beyond what is needed for full functionality. However, as I write this te xt I am struggling to recall any clinical example where this was the case.

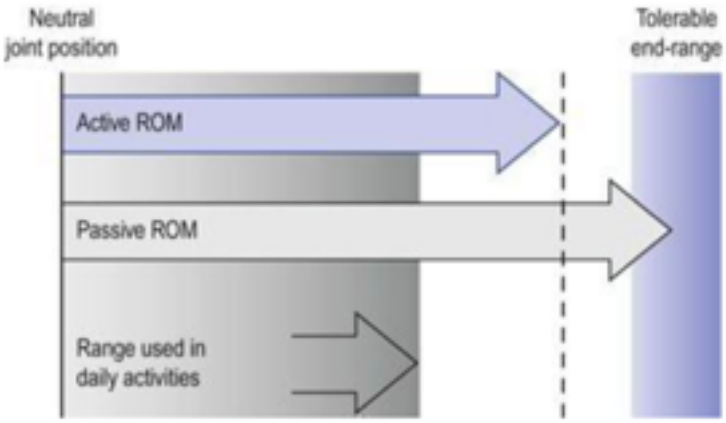


FIGURE 1: Functional ROM
(Range used in daily activities)

## Myofascial Release

Humans have been using soft-tissue manipulation to deal with the fascial system for eons. But when applied regionally, symptomatically, too aggressively, and/or too mechanically, such manipulation simply becomes an attempt to force a system that cannot be forced. It's painful for the client and difficult on the therapist, yielding only temporary results.

As I gained experience with the myofascial system, I found that it responded quite differently from what the earlier research on fascia seemed to show. That research, done on fascia in cadavers, suggested that you couldn't release the three-dimensional web of fascia. I agree that the normal boundaries of the fascial system cannot be altered except surgically, or from the enormous force of trauma,but what earlier research overlooked

was the importance of the ground substance, the gel-like intercellular material in which the cells and fibers of connective tissue are embedded. When exposed to trauma, the ground substance tends to lose its fluidity, and solidifies. I equate it to pouring glue or cement into the interstitial spaces.

It is this dehydration of the tissue,with the accompanying development of cross-links at the nodal points, that can put enormous and excessive pressure on pain- sensitive structures and limit the fascial system's ability to glide. This enormous pressure, approximately 2,000 pounds per square inch, can produce symptoms of pain, including headaches, fibromyalgia, and limited motion. Interestingly enough, myofascial restrictions do not show up in any of the standard tests (X-rays, CAT scans, myelograms, electromyograms), so myofascial restrictions are being completely missed and/or misdiagnosed. Only a portion of the fascial system had been studied by the time of my injury in the 1960s, and it was as if the scientific mind did not understand that comparing living fascia to cadaver fascia is akin to drawing conclusions about trees by studying a telephone pole. Many people are now realizing the ways in which we have been limited by these erroneous assumptions of historical science.[73]

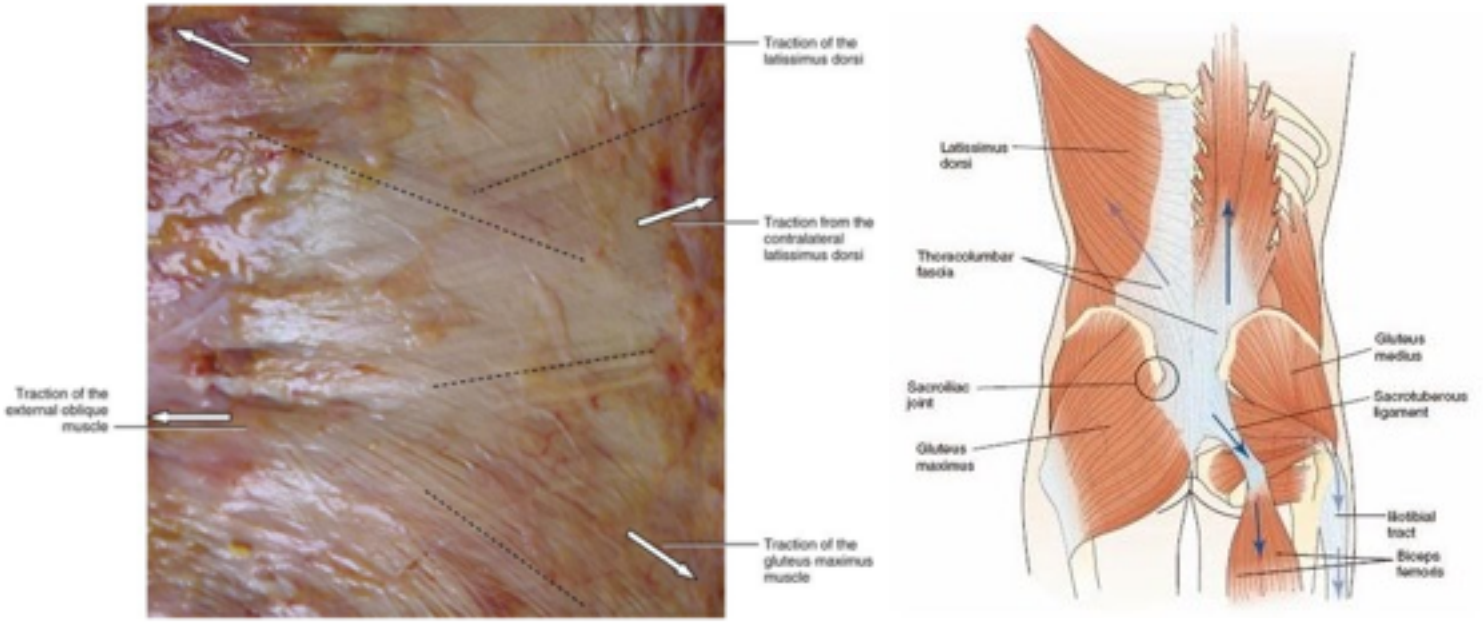


FIGURE 2: A macroscopic view of the posterior layer of thoracolumbar fascia. The multilayered structure is evident. The white arrows show the main direction of muscular contraction and stretching. The dotted lines show the different orientation of the fibrous bundles.

# Methods

## Design and setting

I combine the two major technique into a dynamic myofascial release to treat my client and compare use other technique without DMFR.

## Participants

6 participants(2 males and 4 females) aged 28~40 years, were choose from my clients database which come to my clinic every week and complain stiffness and pain on lower back.

## Procedure

Require patients to stand and I will apply a mild pressure on their lower back to apply my myofascial release and also I will ask patients do the movement as function stretching, to active their muscle and ask them to touch the item that I setting on the ground and ask them to touch item number 1 and move to item number 2 and back to item number 1, and I'm doing the Myofascial Release in the same time, I called this technique as Dynamic Myofascial Release(DMFR), and by asking patients move to another number, and I can feel where is the tight fascial that I need to working around the lower back, when I release all the fascial that I can treat then I will change the location of the item and make the movement more challenged for the patient and do more DMFR to the fascial where need to treat.

I treat my patients that need to receive my massage to ease the muscle tightness or discomfort each week, I start to use the DMFR as a test technique and record the pain scale during to do the functional range of movement(FROM) from the before and after, after 1 week, 2 weeks, and 3 weeks.

# Results

| | Before | After | After 1 Week | After 2 Weeks | After 3 Weeks | Reduce Rate | | | |
|---|---|---|---|---|---|---|---|---|---|
| | B | A | W+1 | W+2 | W+3 | (B-A)B | (B-W +1)B | (B-W +2)B | (B-W +3)B |
| **Patient1 (Male)** | 7 | 4 | 4 | 5 | 5 | 42.9% | 42.9% | 28.6% | 28.6% |
| **Patient2 (Male)** | 7 | 0 | 0 | 1 | 3 | 100.0% | 100.0% | 85.7% | 57.1% |
| **Patient3 (Female)** | 6 | 1 | 0 | 0 | 0 | 83.3% | 100.0% | 100.0% | 100.0% |
| **Patient4 (Female)** | 6 | 2 | 1 | 0 | 0 | 66.7% | 83.3% | 100.0% | 100.0% |
| **Patient5 (Female)** | 5 | 0 | 0 | 0 | 0 | 100.0% | 100.0% | 100.0% | 100.0% |
| **Patient6 (Female)** | 7 | 0 | 2 | 3 | 4 | 100.0% | 71.4% | 57.1% | 42.9% |
| **Average** | 6.33 | 1.17 | 1.17 | 1.50 | 2.00 | 82.1% | 82.9% | 78.6% | 71.4% |

Averagely people who feel the pain level higher than 5 and they will start to looking a treatment to ease the discomfort, by treating the DMFR the pain scale about the lower back during daily movement by out of 10 reduce to 1.17($p<0.05$), a week after 1.17($p<0.05$), 2 weeks after 1.5 ($p<0.05$), 3 weeks after 2.00 ($p<0.05$). And the pain scale reduce rate after the DMFR was 82.1% ($p<0.05$), a week after 82.9% ($p<0.05$), 2 weeks after 78.6% ($p<0.05$), 3 weeks after 71.4% ($p<0.05$).

It works very well, effective rate is over 80%, and the result still working by over 70% after 3 weeks.

# Discussion

1.The DMFR technique that I designed which need more tests and long-term observations to check if it really works well and does it have any side effects, and if there’re any contraindications.

2. For some patients might hard to stand in a fixed position, and some patients may feel annoy need to do lots of movements during Remedial Massage Treatment.

3.Need to find more comparison groups to test the result how much different with Relaxation, Deep Tissue, Myofascial Release, and Functional Stretch, etc.

4.Patients might feel confusing when I ask their pain scale is 1, 2, 3, or 4 without comparison, but they can feel pain-free for a least 3 weeks, and that’s my main target, and trying to improve this technique to make it more effective in different area.

# Conclusions

1.There are significant result in majority data in this study, the patient 1 who work in the farm with heavy duty job average 60 hours a week. The patient 1 keep overloading and using the muscle by repetitive movement and keep dehydrate for a long time because it's not easy to go toilet during the work, he choose to ignore the hydrate as possible as he can. And the long-term dehydrate will make our fascial tight easily[73].

2.I require the patients to keep doing the functional stretch, that can possible to make the patient feel better after a week than the treating day.

# Recommendations

1. The study should be replicated with utilize larger group size and expand the field of criteria by looking at groups other than small groups in the study.
2. Pair up with a personal trainer to guide the patients how to move to help process the Dynamic Myofascial Release, to approach better result.

# Acknowledgments

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Thanks for my wife, Venus always be my first patient to test the every new technique I learned or I designed to reach the goal.

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