

# Fibromyalgia and Chronic Myofascial Pain & Dysfunction: Primary Care Professionals

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There are many varieties of medical primary care providers. They usually share the combination of overwork and information overload. There is a lot of misinformation about fibromyalgia (FM), and a general lack of information about chronic myofascial pain and dysfunction (CMPD) due to trigger points (TrPs). Much of the research hasn't reached clinicians in the front lines. The validity of the reality of fibromyalgia has no place in an expert assessment.”(51) FM and CMPD are real. We don't know all their mechanisms, but we don't know the mechanisms for MS or Alzheimer's either. FM can be found in infants, toddlers, middle-aged, seniors, men, woman, and in any socio-economic group or race. Secondary gain has nothing to do with FM. (60) FM is not autoimmune. (2) We've known that FM is not a muscle disorder, (46) yet a review of FM papers indicates that much research still starts with that misconception. Research describes the essential center of FM dysfunction is in the central nervous system (CNS); the brain and spinal cord. (4; 9; 29; 39; 50 p17; 55) There is a simple 2 page test for the central sensitivity state, (40), although many of the questions may be indicative of TrPs. In FM, the neuroendocrine system may be severely out of balance, and it may take considerable time to restore that balance.

There is a misconception that FM patients are intolerant to pain. “Sensitivity to pain” and “pain tolerance” are different. FM patients have hyperalgesia; a heightened sensitivity toward pain, but may have a greater tolerance to pain. They endure more pain than is often recognized. (37) One of the most important concepts in FM and any central sensitization state is wind-up, or temporal summation of second pain (TSSP). Patients with FM may respond to lower stimuli to maintain a state of central sensitization. (57) Myofascial TrPs that would not cause central sensitization in healthy individuals may be sufficient to maintain central sensitization in patients with FM. (56) TSSP and other concepts are explained in depth on [www.fmcmpd.org](http://www.fmcmpd.org) FM is not progressive (61) If symptoms are getting worse with time, there is some perpetuating factor or a co-existing condition has not been properly addressed. FM tender points were not originally designed as a diagnostic, but only to screen patients for any FM clinical study. Tender point numbers can vary from day to day and hour to hour, just like the symptoms. It is the evidence of central sensitization, with allodynia and hyperalgesia that are important in making the FM diagnosis. (57)

Fibromyalgia can cause diffuse widespread pain, but it does not cause localized symptoms, including numbness and tingling. It's the TrPs that entrap nerves as well as blood and lymph vessels. FM is not a diagnosis of exclusion, although that is also a common misconception. Much

research on FM has been done without regard to co-existing TrPs and their other common perpetuating factors. FM is a pain amplifier, TrPs, arthritis, and other pain sources are pain generators. There is no such thing as a “fibromyalgia trigger point”. Tender points of FM and TrPs of CMPD are different, and in significant ways, even though they may exist in the same patient. (17; 38; 50 p 18) Failure on the part of care providers to recognize myofascial TrPs often leads to costly and unnecessary tests and procedures. This failure to diagnose can result in harm to the patients. (37) Researchers are beginning to document that FM patients often have multiple co-existing conditions. (5; 45) What many may not yet understand is that many of these conditions are interactive, and can be utilized as avenues to treat the central sensitization of FM.

So far, many genes have been implicated in the genetic tendency to develop FM. (1; 24; 36) It then takes insult to the CNS, whether it be repetitive motions, static postures, trauma, infection, exposure to toxins, or other initiating factor to start the TSSP process. One factor that can do this is lack of restorative sleep. (23) Sleep dysfunction can cause increase in FM pain and associated depression. (6) Research indicates that FM fatigue is associated with lack of restorative sleep, (33) and the first step in treatment of any patient with both fatigue and pain that is chronic is a good, in-lab sleep study.(41; 52) It is an error to check for sleep apnea alone. One must check for associated sleep dysfunctions including abnormal sleep architecture. Ensure that sleep is not fragmented, and that the patient gets adequate quality sleep stages in their proper intervals.

The cognitive deficits of FM can be more disabling than the pain, though they interact. The brain can be so preoccupied with processing pain signals that it cannot function. (20) Changes in oscillatory activity and impaired connectivity have been implicated in FM attentional deficits. (21) There is greater lag time to access words stored in long-term memory, (26) and research shows auditory and pressure sensitivity suggesting global amplification of sensory input. (14) A specific scale has been developed to measure what patients call “fibrofog”.(27) “Chronic myofascial pain, caused by myofascial trigger points, is associated with localized brain atrophy in areas involved in pain processing and modulation, among others”. (34) There are also specific TrPs that can cause loss of attention and focus (rectus capitis anterior, lateral), disorientation or confusion (sternal SCM). Cognitive deficits are often due to more than FM. We can treat the contributors of cognitive dysfunction that we know are treatable, and then more easily deal with what remains.

The central sensitization of FM may be caused by many initiating factors including infections, trauma, severe pain or chronic pain, sustained grief, or other conditions that may contribute to the initiation of TrPs. Several researchers have found that controlling TrPs minimize FM central sensitization. (7; 10; 13; 30; 54; 62) One paper recommends “assessment and treatment of concurrent TrPs in FMS should be systematically performed before any specific fibromyalgia therapy is undertaken” and addresses the role TrPs play in other pain conditions such as headaches and visceral diseases. (19) We have long known that TrPs can cause pain as severe or more severe than other causes, (59) and even a few TrPs can cause “agonizing incapacitating pain” (50 pg 13) Reflect on the pain level of a patient with hundreds of TrPs, amplified by FM. You can see how one cannot adequately treat patients with one of these conditions without

recognizing and understanding the other. It is the combination of pain generators such as TrPs and pain amplification of FM that causes the extreme disabling pain.

This is the TrP cycle as we presently understand it: Mechanical stress (overload) initiates excessive acetylcholine release, which causes excessive resting calcium ion release from the sarcoplasmic reticulum. This causes multiple contraction knots. Contraction knots are directly responsible for the palpable TrP nodule. The taut band is primarily responsible for the increased muscle tension and for the shortened rest position of the muscle. In one myofascial TrP there may be multiple contraction knots. The endplate dysfunction characteristic of TrPs involves both nerve terminal and postjunctional muscle fiber. This relationship identifies TrPs as a “neuromuscular disease”. (47) Reflex inhibition causes the local energy crisis to develop from increased energy demand and decreased energy supply. This results in pain, motor and autonomic effects. Sensitization of local autonomic fibers causes increased acetylcholine release, which causes the release of calcium ions, which continues the cycle. The pH drop at the TrP site alone is sufficient to cause a change in the nociceptive milieu, and the addition of pro-inflammatory mediators such as substance P, bradykinin and cytokines may aggravate this change. (18) “Myofascial pain... often exists as part of a clinical complex that includes these other soft tissue conditions, i.e., it is not a diagnosis of exclusion.” (3, 16) Once you become familiar with the concepts of myofascial medicine, you will be amazed at their ubiquity in your practice.

Sarcomeres are the smallest functional contractile unit of skeletal muscle. The TrP sarcomere has been verified by muscle biopsy. (49) TrPs have been imaged at the National Institutes of Health USA, (44) and TrP taut bands have been imaged at the Mayo Clinic USA. (8) There is no excuse to doubt their existence. Over 30 specific chemicals released into interstitial fluid during a TrP local twitch response have been sampled and identified, and cause a significant acidification of the surrounding tissue.(43) TrP may be active or latent. Latent TrPs don't cause spontaneous pain, but restrict movement, can cause muscle weakness, autonomic and proprioceptor concomitants and other symptoms. Active TrPs also cause spontaneous pain. Be gentle when palpating for TrPs. Pressing TrPs can activate them and cause pain for days or even weeks. Extra medication including muscle relaxers and pain meds may be required before and after an exam, and your patient may need extra supportive therapies such as spray and stretch, TrP myotherapies (not the same as myofascial release), and craniosacral release. Patients with CMPD may have fibrotic muscles, or muscles swollen with interstitial infiltrates; you may be unable to palpate the TrPs or tender points, or even to feel the ropy bands. In those cases, go by the associated specific TrP symptoms, pain referral patterns, and pain at the end of range of motion. Often, it is easier to palpate ropy bands in limbs if the limb is extended three-quarters of the way, and the same is true of other extensible areas such as the jaw. Once galvanic muscle stimulation, spray and stretch, TrP injection, dry needling, or other physical therapy modalities have been used and all the perpetuating factors have been brought under control, you will be able to palpate the TrPs.

Help for history taking, exam and treatment of patients with FM and CMPD is available, (53) and your patient education tasks will be easier once that book is in their hands. There are in-depth diagrams and directions for TrP injection in the definitive myofascial medical texts (50, 58), and it is appropriate to open these books and refer to them while palpating, positioning, injecting, or otherwise treating your patients. The diagrams in those books are often misunderstood, as the “X” marks were intended by the authors as **guidelines** only. TrPs can occur in any part of the muscle and its attachments. TrPs can also occur in other tissues. There are many therapies for TrPs. On the website [www.fmcmpd.org/](http://www.fmcmpd.org/) you will find a CMP chapter explaining Stage I CMP (without central sensitization) and Stage II (with central sensitization). I use the term **CMPD** to recognize the dysfunctions caused by TrPs, as they can be disabling as the pain and are often overlooked. CMPD is too often called myofascial pain syndrome (MPS), but so is temporomandibular disorder and other jaw pain, leading to massive diagnostic and research confusion. (48) Consider papers indicating a jaw splint to be one of the most effective treatments for MPS. How much can that mouth splint help your chronic pelvic pain patients? See the problem?

TrP injection or needling is effective if the proper procedure is utilized, although identifying and correcting perpetuating factors is also necessary. (16) A thorough knowledge and understanding of TrP injection technique, including palpation, positioning, and proper injection procedure is vital. Training is available ([www.myopainseminars.com](http://www.myopainseminars.com)) Research indicates that steroids add nothing to the injection, (32) and that the least myotoxic [www.myopainseminars.com](http://www.myopainseminars.com) agent such as procaine or lidocaine (without epinephrine) should be used. (63) It is the needle that breaks up the TrP, and not the injectate. (42)

Be careful when recommending exercises and physical therapies, Muscle weakness may be due to dysfunctional reciprocal inhibition. A specific muscle with TrPs may work well during one action but can be inhibited during another. (50 p 27) The inhibiting muscle must be identified and its TrPs released, and the affected muscles must be lengthened and returned to their normal suppleness. After treating the inhibiting muscle TrPs, the muscle dysfunction will persist until the muscle is retrained. **You cannot strengthen a muscle with a trigger point.** A muscle with TrPs is already working as hard as it can work. It is physiologically contracted, and that is not the same as contraction. When a muscle is in sustained contracture, there is “increased metabolic demand and impaired metabolic supply.” (50 p 71). The last thing needed by a contracted muscle is more work. Trying to strengthen a muscle with TrPs can cause the TrPs to worsen and develop satellite and secondary TrPs. Inappropriate exercise is a preventable perpetuating factors. Patients often have clusters or chains of TrPs, and clinicians need to be on the alert that when one TrP is present in a patient with chronic symptoms (not always pain) the TrPs can cause multiple dysfunctions including weakness, as well as other symptoms, before they cause pain. The sustained muscle shortening of TrPs can literally have a crushing effect on microcirculation.

You need the right dose, the right timing, and the right kind of exercise, and these change as your patient improves or has additional setbacks. You can't rely on a PT knowing that treating patients with FM and CMPD is not the same as treating a patient with a few TrPs from sports injuries. Too often they are taught to strengthen. TrPs interact, and FM amplifies the pain and dysfunction. It's not uncommon for patients to feel nausea or a dramatic increase in muscle aches, especially headaches, and/or exhaustion after any TrP therapy moves a large amount of toxins and wastes from their myofascia. Warn patients to rest after bodywork and other therapies. As TrPs are inactivated, the patient should begin carefully graded but nonrepetitious exercises in sets, to eventually increase strength and endurance. This process can't be rushed. Work with a good bodyworker; ideally, a trained TrP therapist who is knowledgeable about both FM and CMPD. Initially, the patient may be able to tolerate only one short session once a week. The body needs time to process released toxins and waste. Continuing to exercise in spite of pain simply aggravates the abnormal pain filters. Prescribing exercise should be considered as carefully as writing a prescription. It must be tailored to the needs of the individual patient. (35) Patients with the combination of FM and CMPD may have metabolic irregularities which cause them to become exhausted from the slightest exertions. This means that their exercise programs must begin very conservatively, and care must be taken to ensure that they recover between exercise sessions.

I have seen too many patients with active TrPs who were put into weight-training and work-hardening programs. These programs often caused total disability. Patients with TrPs cannot take repetitions, and their muscles cannot be strengthened until the TrPs are gone (50). Patients with CMPD often exhibit Delayed Inset Muscle Soreness (DOMS). They cannot gauge how much exercise will hurt them until the following day, or even the day after. During the stage in which healthy muscle is relaxed, a muscle with TrPs does not relax. (22) This contributes to muscle fatigue. The contracture of muscle with TrPs muscle can maintain displacement stress on nearby joints, and the abnormal sensory input from the joint can affect the muscle (TPM P 40) this leads to bidirectional interaction with arthritic conditions, including degenerative joint disease.

When the myofascial nature of pain is unrecognized, such as the pain caused by TrPs in the pectoral muscles that mimics cardiac pain, the symptoms are likely to be diagnosed as neurotic, psychogenic, or behavioral. This adds frustration and self-doubt to the patient's misery and blocks appropriate diagnosis and treatment (50 p14). FM and CMPD interact, but once the concepts of FM and CMPD are understood, your diagnostic skills will sharpen and you will more easily be able to separate the two conditions. Case reports indicate that a well-educated and function-oriented patient coupled with a care provider who is well-trained in the recognition of FM and TrPs can work as a team to significantly improve the patient's quality of life, improving function and decreasing pain level.(11)

The secret to managing FM is to identify the pain generators, including TrPs, and treat and control them, and identify and eliminate the perpetuating factors if possible, including co-

existing conditions. If the perpetuating factors cannot be eliminated, they must be managed to minimize their impact on the patient's symptom burden. Anything that initiates, aggravates or perpetuates a TrP is called a perpetuating factor, and the concept of perpetuating factors works with any medical condition. There is a very detailed chapter concerning perpetuating factors in Vol. 1 of the Trigger Point Manual (50) Chronic pain cannot be managed with cookbook medicine. It takes **time**. Take the time to talk with your patient, and to listen. If you ask the right questions, you'll find out what you need to know. You will both be rewarded with the improvement your patient will show, and time will be saved in the long-run. It isn't a smooth road. There are plateaus, and there are setbacks. Sometimes flare or another stressor may cause the need to temporarily increase meds and bodywork and mindwork. That need should end as the stressor is relieved. It is typical for these illnesses to have symptom variation from hour to hour and day to day. This is part of the difficulty dealing with them, for both you and for your patient. It may also be difficult for your patient's family and companions and family to understand. Ensure that your patient understands how to build a good support structure.

Anything that can cause noxious stimuli to the already hypersensitized CNS can worsen FM, and that includes pain itself. FM flare is a period wherein symptoms increase in severity, the CNS is on a hair trigger, the patient is in sensory overload, and new symptoms can appear. FM flare may be due to activation of multiple latent TrPs, causing heightened CNS sensitivity. FM and CMP pain can be all-consuming. Even though the patient may look fine, many patients have been driven to suicide. (28; 12). Pain medication should be given with the goal of return to maximum function with the least possible pain. Believe that your patients hurt as much as they say they do. You can't see the pain. A VAS 1-10 scale is only good in comparing the present pain to what that patient has experienced before. There is danger in the fear-based action of trying to eliminate opioids from our pain control tool chest. (25) "Chronic pain patients who are on successful opioid treatment should not be unilaterally compelled to wean off opioids." These authors are authorities. Dr. Kronke is a professor at the University of Indiana, School of Medicine, and a research scientist. Dr. Cheville is chair of research at the Mayo Clinic Dept. of Physical Medicine and Rehabilitation. Don't remove successful pain treatment unless you have removed the pain.

Remember that myofascial TrPs are the great mimic. They can cause pain and other altered sensations, such as numbness and itch, but they can also cause dysfunction such as buckling knee and ankle, retraction of the testicle, erectile dysfunction, dyspareunia, intestinal cramping, menstrual pain, blurring of vision, dizziness, and loss of focus. Some cause symptoms that seem neurological. TrPs are not an either/or situation. You may have a viscerosomatic or somatovisceral interaction putting your patient in a negative spiral. The key is always to find the (initiating, aggravating, perpetuating) perpetuating factors. These may be metabolic, such as insulin resistance or thyroid resistance (they are on the same gene), lack of restorative sleep (which may itself have multiple perpetuating factors), structural, or behavioral, such as smoking, poor posture, or poor diet. Visits should be about empowering patients to make the wisest and

most efficient choices so that they can regain as much function as possible, with the highest quality of life. They cannot do this alone. They need your help find co-existing conditions that are perpetuating factors, such as Ehlers-Danlos Syndrome, Leaky Gut, vitamin and mineral insufficiencies.

Depression is often a result of chronic pain. This is especially true when patients have had a long period of undiagnosed illness, or when their doctors and family have repeatedly denied the reality of their illnesses. Some patients do have emotional or mental problems, and these can worsen the physical symptoms of FM and CMP. Often, the constant effort of dealing with their so-called “untreatable pain” has reduced physical activity, limited social activities, impaired sleep, caused loss or change of family role, and perhaps been responsible for the loss of a job. When patients mistakenly believe that they must live with and endure undiagnosed TrP pain, they feel hopeless and helpless. Their TrPs become latent. Latent TrPs are land mines waiting for activation, which can come from a fall or infection or any other stressor. Patients must learn that TrPs are responsive to treatment. Patients must learn that perpetuating factors of both FM and CMP can be brought under control. Conversations with patients should be about empowerment, not invalidation.

The Trigger Point Manuals should be on your desk and well-used. They are a great investment for your value as a medical practitioner, and your patients’ quality of life. Refer to them often. Do not just look at the diagrams. Much diagnostic treasure is in the text. Nowhere else can you get such a return on your time. The International Myopain Society and the Myopain Seminars are excellent resources for medical care providers. The National Association of Myofascial Trigger Point Therapists has an exceedingly useful website ([www.myofascialtherapy.org](http://www.myofascialtherapy.org)), and you would do well to recruit one of these specialists to work in your practice. There are resources specifically written for patients with FM and CMPD and their medical professionals (53; [www.fmcmpd.org](http://www.fmcmpd.org)). Treating patients with both FMS and CMP can be a “throw away your crutches and walk” kind of experience that is all too rare in a medical professional’s life. It is not that unusual to have one of these undiagnosed or misdiagnosed patients come bed-bound or in a wheelchair, and it is in your power to get that patient walking, functioning, and managing his or her own medical care, and enjoying life once again.

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