

What Primary Care Physicians, Rheumatologists and Physiatrists Should Know about FMS and CMP

By Devin J. Starlanyl

This information may be freely copied and distributed only if unaltered,
with complete original content, including: © Devin Starlanyl, 1995-2002.

Please read "What Every Health Care Provider Should Know About FMS and CMP."

Who Gets Fibromyalgia or Chronic Myofascial Pain?

People of any age, race or gender can get FMS or CMP. Infant colic is often caused by trigger points (TrPs) that can be relieved with gentle Spray and Stretch and Cranio-Sacral Release. Fibromyalgia in youth may start with a flu-like illness and then manifests as "growing pains," which can also be caused by TrPs. Fibromyalgia (FMS) is the commonest cause of widespread pain (Bennett, 1995), yet it usually remains undiagnosed for a long time. Uncertainty and frequent misdiagnosis can cause considerable havoc in the lives of patients. Men do get FMS, and they are not immune to the doubt and lack of support with which an invisible illness is met.

There is no gender bias in chronic myofascial pain from trigger points (TrPs). Patients can have both FMS and CMP, but each condition must be treated differently. The key is always to find the perpetuating factors. These may be metabolic, such as insulin resistance, sleep fragmentation or thyroid resistance, structural (myofascial TrPs can perpetuate the central sensitization of FMS), or behavioral, such as smoking, poor posture, or poor diet. Once you find the right combination of medications, physical therapy, and coping skills, your patient can avoid many of the absences from work or school that would otherwise occur.

They're Real

At the Travell Focus on Pain Seminar 2000, I. Jon Russell MD, the editor of the Journal of Musculoskeletal Pain, mentioned the use of the Functional MRI. This imaging device shows the brain in action. He told how, in a healthy individual, when you pressed on a tender point site, there was a minimal response, but in a patient with FMS, "...the result was wild – the whole brain went crazy". Those Functional MRIs are documentation that something is happening to people with FMS that doesn't happen to others. FMS can be as disabling as rheumatoid arthritis, and about 30% of FMS patients cannot continue in their same occupations due to their chronic, unrelenting symptoms (Wolfe, 1989). "It is now firmly established that a central nervous system (CNS) dysfunction is primarily responsible for the increased pain sensitivity of fibromyalgia" (Simons, Travell and Simons, 1999 p 17). There is a generalized CNS-mediated deep tissue sensitivity in FMS that includes the muscles, which is why so many people mistakenly believe that it is a muscular condition. It is not. (Simms, 1998) "The most aggressive challenges of the FMS concept have been from legal defenses of insurance carriers motivated by economic concerns. Other forms of critique have presented as psychiatric dogma, uninformed posturing, suspicion of malingering, ignorance of nociceptive physiology, and occasionally have resulted from honest misunderstanding." (Rau, Russell 2000) These patients are often in dire need, and they look to you to fill some of those needs. Their visits should be about empowering them to make the wisest and most efficient choices so that they can regain as much function as possible, with the highest quality of life.

Anything that results in tissue injury, whether from more obvious physical trauma such as an auto accident or from more subtle biochemical damage, can cause hypersensitivity at the site of the injury. If there is repeated or continued trauma, other areas may develop the hypersensitivity (Yaksh, Hua, Kalcheva et al. 1999). This can lead to a state of central sensitization. Fibromyalgia is not just achy muscles. It is a multisystem illness, and may be present in addition to other illnesses (Martinez-Lavin 2001). The neuroendocrine system may be severely out of balance, and it may take a great deal of time to restore that balance.

Fibromyalgia is not the same as CFIDS, although they are both probably part of the same family of neuroendocrine dysfunctions. In one study, levels of substance P were determined in the cerebrospinal fluid in 15 patients with CFIDS. All values were within normal range. The majority of patients with FM have increased substance P values in the cerebrospinal fluid. The results of this study support the notion that FMS and CFIDS are different disorders in spite of overlapping symptoms (Evingard, Nilsson, Lindh, et al. 1998). Another study points out that "In FMS, there is a condition of physiological hyperarousal. In CFIDS, a blunted response, the exact opposite, occurs" (Crofford, 1998).

Fibromyalgia is not progressive (Wolfe, Anderson, Harkness et al. 1997). If the symptoms are getting significantly worse with time, that signifies there is some perpetuating or aggravating factor or a co-existing condition that has not been properly addressed. If you identify it and deal with it thoroughly and promptly, the symptoms may ease considerably. Fibromyalgia is not the same as chronic myofascial pain (Gerwin, 1999). It is fundamentally different in an important way (Simons, Travell and Simons, 1999 p 18.) *There is no such thing as a fibromyalgia trigger point.*

A myofascial TrP is always found in a taut band, which is structurally related to contraction knots caused by a *thousand-fold* increase in the release of acetylcholine, an important neurotransmitter. This action takes place in an area of the muscle where nerves end, which is called a motor endplate (Gerwin, 1999). The cause of TrPs appears to involve serious disturbances of the nerve ending, as well as dysfunction of the contractile mechanism at multiple dysfunctional endplates (Hong, 1999).

For many years, the emphasis has been on the pain caused by myofascial TrPs, because the pain can be so prominent. Even more significant, however, is the increased tension of the muscle caused by the TrP, and the muscle dysfunction the TrP causes. *TrPs cause muscle weakness and restricted range of motion.* Physical and occupational therapists, as well as doctors, often recommend strengthening exercises, without understanding that the TrP is inhibiting the muscle, and ***you cannot strengthen a muscle with a trigger point.*** Trying to strengthen a muscle that harbors TrPs will cause the TrPs to worsen and develop satellite and secondary TrPs, and your patient can become disabled. Improper physical therapy and lack of adequately trained medical care providers can be prime perpetuating factors.

Myofascial TrPs can be identified **and documented** electrophysiologically by characteristic *spontaneous electrical activity* (SEA) (Hubbard and Berkoff, 1993). There are also ultrasound images of a TrP local twitch response, and biopsies of myofascial TrPs that show the contraction knots and giant rounded muscle fibers. "The endplate dysfunction characteristic of MTrPs involves both the nerve terminal and the postjunctional muscle fiber. This relationship identifies MTrPs as a neuromuscular disease." (Simons, 1999). They may also

be identified histologically by *contraction knots* — the palpable lumps you find on examination.

At the Focus on Pain Seminar 2000, David Simons explained 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, with the area of most intense shortening of the sarcomeres occurring in the region of a motor endplate. Contraction knots are directly responsible for the palpable nodule at the myofascial TrP and for the palpable taut band. 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 are many contraction knots. Contraction knots are usually about 100 micrometers in diameter, which is twice the diameter of a normal muscle fiber. 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.

Patients with specific patterns of referred pain (TrPs), indications of blood vessel, lymph, or nerve entrapment or proprioceptive disturbances should be evaluated for myofascial TrPs. If you can palpate lumps and ropy bands, look for specific myofascial referred TrP pain patterns. If your patients have muscles that have become fibrotic, you may not be able to palpate the TrPs and taut bands, but a good history will tell you where the TrPs are. "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" (Simons, Travell and Simons, 1999, p14).

Once you become familiar with the concepts behind the diagnoses of FMS and CMP, your diagnostic skills will become sharper and you will more easily be able to separate the two conditions, even when they co-exist in the same patient. It is important to understand the differences, as this changes treatment strategies (Borg-Stein and Stein 1996).

FMS Tender Points

These tender points are often found in the official "eighteen locations." In cases of traumatic FMS, they may be clustered near trauma sites (including degenerative changes such as those from OA or DDD, or surgical incisions). Muhammad Yunus, M.D., has found that, when testing for tender points of fibromyalgia, a good indicator of proper pressure is that when you use your thumb to press, you notice a blanching of the fingernail. This produces 4 kg of pressure (Yunus 1988). The tender points were not designed as a diagnostic, but only to screen patients for any FMS 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 diagnosis (Staud, Vierck, Cannon et al 2001).

CMP Trigger Points

Trigger points may be active or latent. Latent TrPs restrict movement and cause muscle weakness, but don't cause pain until they are activated by immobility, stress, or many

other possible perpetuating factors. Be gentle when checking for TrPs. Pressing TrPs can activate them and cause extreme pain for days or even weeks. Extra medication before and after an exam may be required, and those muscles tested may need myotherapy treatment such as spray and stretch. Individual TrPs can be difficult to live with, but they may respond immediately to specific therapy.

It is not unusual in cases of CMP for the muscles to feel as hard as cement. You may be unable to palpate the TrPs or tender points, or even to feel the ropy bands. Often, it is easier to palpate ropy bands in the limbs if the limb is extended three-quarters of the way. Once galvanic muscle stimulation, Spray and Stretch, and other physical therapy modalities have been used to break up the myofascial splinting, and all the perpetuating factors have been brought under control, you will be able to palpate the TrPs. Inability to diagnose myofascial pain "...often leads to over-investigation, unnecessary medical intervention, and iatrogenic harm with serious cost implications." (Meyer 2002)

Perpetuating Factors

Fibromyalgia and chronic myofascial TrPs often perpetuate each other. Anything that can cause pain or other strong stimuli to the already hypersensitized central nervous system can aggravate FMS. If the aggravation is strong enough or repetitive enough, flare may develop. Fibromyalgia flare is a period wherein symptoms increase in severity, the central nervous system is on a hair trigger, and new symptoms can appear. The best way to deal with flare is to prevent it. In "The Fibromyalgia Advocate" and "Fibromyalgia and Chronic Myofascial Pain: A Survival Manual 2nd edition", there are detailed instructions to help your patient to avoid flare, and help him or her though it if it comes.

If an altered HPA axis, altered metabolism, and reactive hypoglycemia or insulin resistance have conspired to create abdominal obesity, or if there is extreme muscle guarding, rely on your patient's history to give you an indication of the pattern of possible TrPs. A careful history is the most important part of the physical exam, but you need to gain experience palpating for TrPs. Do understand that palpating TrPs can activate them unless you use a very light touch. Check for perpetuating factors. This often takes a bit of detective work, and the help of many other medical team members. Identifying and dealing with the factors that are perpetuating fibromyalgia and myofascial pain symptoms is the key to better health for your patient. The most common perpetuating factors of TrPs are the following conditions: fibromyalgia, impaired sleep, reactive hypoglycemia, paradoxical breathing, hypothyroid, postural dysfunction, skeletal asymmetry and disproportion. Also consider nutritional inadequacies, anything that impairs muscle metabolism, exposure to toxins, chronic infections, psychological factors, allergies, inappropriate physical therapy, and stress. Considering that FMS may involve multiple imbalances in neuroendocrine hormones, it may take a lot of patience to sort everything out and help your patient to regain health. Consider that chronic autonomic stimulation, as often occurs in FMS, is a cardiovascular risk factor, so do not take FMS lightly (Curtis, O'Keefe Jr 2002). Glucose metabolism is also under neuroendocrine control, so consider metabolic syndrome as a possible perpetuating factor (Peters, Schweiger, Fruwald-Schultes et al 2002).

Exercise

I have seen too many patients with active TrPs who were put into weight-training and work-hardening programs. With the best of intentions, these programs often transformed them into totally disabled people. Patients with TrPs cannot take any repetitions, and their

muscles cannot be strengthened until the TrPs are *gone* (Simons, Travell, Simons 1999). Patients with FMS 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 are recovering between exercise sessions. Listen to your patient. Because of their healthy appearance, FMS patients are frequently denied the support that other handicapped patients take for granted. Set realistic vocational, social, and recreational goals.

Prescribing exercise should be considered as carefully as writing a prescription. You need the right dose, the right timing, and the right kind. Do not rely on the PT degree to ensure that a bodyworker is trained in treating patients with both FMS and CMP. It's not uncommon for patients to feel nausea or a dramatic increase in muscle aches, especially headaches, and/or exhaustion, after any physical therapy has moved a large amount of toxins and wastes from their constricted muscles. Warn patients to take it easy after bodywork and other therapies. As TrPs are inactivated, the patient should begin carefully graded but nonrepetitious exercises to increase strength and endurance. You need to work with a good bodyworker who is knowledgeable about both FMS and CMP. They must understand that you cannot strengthen a muscle harboring a TrP. Initially, the patient may be able to tolerate only one short bodywork session once a week. The body needs time to process released toxins and waste. There is good evidence that continuing to exercise in spite of pain simply aggravates the abnormal pain filter (Bennett 1997).

Pain

The combination of FMS and CMP is not to be taken lightly. Neither are individual TrPs. "A swimmer may drown from a muscle cramp produced by a myofascial trigger point. Myofascial pain has driven some patients to suicide. Myofascial back pain is a major, unrecognized source of industrial disability" (Travell and Simons, 1983, p18). Fibromyalgia amplifies the pain and other symptoms of myofascial TrPs. Consider the new understanding of central nervous system sensitization when prescribing medications and treatment regimens (Lidbeck 2002). Some forms of bodywork and mindwork may be very helpful for both FMS and CMP (Starlanyl, Copeland 2001). Pain medication should be given with the goal of return to function with the least possible pain. Research shows that the increasing use of opioids to treat pain has not caused a corresponding increase in opioid abuse (Joranson, Ryan et al 2000).

Believe that your patients hurt as much as they say they do. You can't see the pain, and a 1-10 scale is only good in comparing the present pain to what that patient has experienced before. Undiagnosed FMS and CMP pain can be all-consuming, and even though the patient may look fine, many patients have been driven to suicide. Chronic pain can kill (Liebeskind 1991; Fishbain, Goldberg, Rosomoff, et al. 1991).

Psychological Information

Fibromyalgia is not a mental illness, and must not be categorized as such. Studies have shown that the incidence of mental problems is no higher with FMS patients than with any other type of chronic pain syndrome (Goldenberg, 1989; Merskey, 1989). "There is now clinical evidence that FMS represents a distinct rheumatic disorder and should not be regarded as a somatic illness secondary to psychiatric disorder" (Dunne and Dunne 1995). Psychiatric Diagnostic Interview data failed to discriminate in any major way between primary fibromyalgia syndrome and rheumatoid arthritis (Ahles, Khan, Yunus et

al. 1991).

Your patients may become confused or extremely stressed during office visits. Some researchers have found that FMS causes a slow rate of information-processing or slowed psychomotor speed in tasks that require sustained effort (Landro, Stiles, Sletvold, 1997). Cognitive complaints are common in many chronic pain states. Some researchers feel that these may be due to an interference between ongoing pain and mental tasks, as they share common and limited attentional resources (Grisart and Plaghki, 1999). Pain processing eats up our thought processing networks, thereby interfering with concurrent cognitive tasks such as thinking, reasoning, and remembering (Kuhajda, Thorn and Klinger, 1998). Inability to make a simple decision may be keeping your patient from living a normal life. This lack of decisiveness may be caused by adrenalin and noradrenalin imbalance.

People with FMS do not have the same mental agility as healthy people of the same age. They also experience significant vocabulary deficits (Glass, Park 2001). In one study, FMS patients reported lower memory capacity, less control over memory function, more cognitive deficits and greater memory deterioration than a group of healthy controls or like-education who were 20 years older. This study showed that the FMS patients were accurate in their assessment (Glass, Park, Crofford 1999).

Depression

Depression is often a result of chronic pain syndromes. This is especially true when patients have had a long period of undiagnosed illness, or when their doctors and family have repeatedly said to them, "It's all in your head." Of course, it is also true that some patients do have emotional problems, and these can worsen the physical symptoms of FMS 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.

Acute pain that diminishes in the course of a natural healing process is generally manageable psychologically, but recurrent or persistent pain caused by an unrecognized cause threatens future function and well-being. It can lead to frustration, depression, and progressive disability. When patients mistakenly believe that they must live with and endure undiagnosed TrP pain because they have been told it is arthritis or that the cause is unknown, they restrict their activities to avoid the pain. Their TrPs become latent, and latent TrPs are time bombs waiting for activation, which can come from a fall or an infection or any other stressor. Patients must learn that TrPs are responsive to proper treatment. Patients must learn that perpetuating factors of both FMS and CMP can be brought under some control. Conversations with patients should be about empowerment, not invalidation.

If relevant, order tests to identify coexisting conditions that might be contributing factors. There is a very detailed chapter concerning perpetuating factors in Vol. 1 of the Trigger Point Manuals (Simons, Travell and Simons). Take the time to talk with your patient. Take the time 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. 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 symp-

tom 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 co-workers to understand. Ensure that your patient understands how to build a good support structure.

Therapies

What eases one TrP may adversely affect another, setting up a rebound contraction. When the whole body, or most of it, is riddled with TrPs, and there is FMS amplifying the picture, untangling the knot is difficult and frustrating. In addition, any TrP may cause proprioceptor dysfunction and autonomic concomitants. TENS units are not the best electrical unit for breaking up trigger points, because they don't create the necessary muscle contraction. Microstim, neuromuscular electrical stimulation, and galvanic stimulation may work well, and there are many varieties. Some home units are available with a prescription.

Trigger point injections can be part of an overall therapy regimen, but they do not stand alone, and they must be done properly. Each muscle must be positioned so that you can palpate all of the TrPs, and you need to treat every muscle in the muscle function group. There may be TrPs in every layer of every muscle. Stretching is an integral part of TrP injection, and "spray and stretch" technique may be very helpful. It can be useful to have several myotherapists with different types of skills working with you on these complex pain patients, but you must still identify and deal with the perpetuating factors. Stretched and injected muscles are likely to be sore for two to three days following treatment. Compression to reduce bleeding, followed by moist heat plus putting the muscle through 3 passive range of motion stretches may help relieve post-injection soreness. Your patient must take it easy during the recovery period. Soaking in a hot bath with Epsom salts and a tablespoon of ground ginger may be soothing. Strenuous activities should be avoided while the muscles are sore, including shopping, gardening, and traveling. Patients with FMS may not find injections as effective as patients with just CMP. The injections may hurt more, and their effects may last for shorter periods of time. See the Travell and Simons Trigger Point Manuals (1992; 1093). Each specific trigger point injection technique is described in detail in those books. If you get a chance to attend a seminar on FMS and/or CMP, do so. I'm sure you will find it worth your time. Contact Robert Gerwin, M.D., for information on Myofascial Pain Seminars at (301) 656-0220 or www.painpoints.com. Dr. Gerwin wrote the chapter on perpetuating factors in the last Trigger Point Manual, and he is an expert in the diagnosis and treatment of myofascial pain.

"Myofascial Pain and Dysfunction: The Trigger Point Manual Volume I and Volume II" (1999; 1992) should be in your office library and in your hospital library. The causes, perpetuators, and remedies for trigger points are all there. Keep the manuals on your desk and refer to them often. Don't just look at the diagrams. Think of your time with them as an investment. Nowhere else can you get such a return on your time and money. The Journal of Musculoskeletal Pain is an excellent source of new information for physicians.

The treatment of 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 physician'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 functioning, managing his or her own medical care, and enjoying life once again. What a deal!

References

- Bennett, R. M. 1995. Fibromyalgia: The commonest cause of widespread pain. *Frontiers* 21(6): 269-275.
- Borg-Stein, J. and J. Stein. 1996. Trigger points and tender points: one and the same? Does injection treatment help? *Rheum Dis Clin N Am* 22 (2): 305–322.
- Consensus Document on Fibromyalgia: The Copenhagen Declaration. 1992. Published in *Lancet*, vol. 340, Sept. 12, 1992, and *J Musculoskel Pain* 1(3/4) 1993.
- Crofford, L. J. 1998. Neuroendocrine findings and patients with fibromyalgia. *J Musculoskel Pain* 6(3): 69
- Curtis B. M., O’Keefe J. H. Jr. 2002. Autonomic tone as a cardiovascular risk factor: the dangers of chronic fight or flight. *Mayo Clin Proc* 77(1): 45-54.
- Evengard, B. C. G. Nilsson, G. Lindh et al. 1998. Chronic fatigue syndrome differs from fibromyalgia. *Pain* 78(2): 153-155.
- Fishbain, D. A., M. Goldberg, R. S. Rosomoff and H. Rosomoff. 1991. Completed suicide in chronic pain. *Clin J Pain* 7(1): 29–36.
- Gerwin, R. D. 1999. Differential diagnosis of myofascial pain syndrome and fibromyalgia. *J Musculoskel Pain* 7(1-2): 209-215.
- Glass, J. M., D. C. Park. 2001. Cognitive dysfunction in fibromyalgia. *Curr Rheumatol Rep* 3(2): 123-127.
- Glass, J. M., D. C. Park, L. J. Crofford. 1999. Metamemory in fibromyalgia. Poster Presentation at Am Col Rheumatol 63rd Ann Sci Meeting Hynes Convention Center Boston MA Nov 13-17.
- Goldenberg, D. L. 1989. Psychological symptoms and psychiatric diagnosis in patients with fibromyalgia. *J Rheumatol Suppl* 19: 127-130.
- Grisart, J. M. L. H. Plaghki. 1999. Impaired selective attention in chronic pain patients. *Eur J Pain* 3(4): 325-333.
- Hong, C-Z, 1999. Current research on myofascial trigger points: pathophysiological studies. *J Musculoskel Pain* 7(1-2): 121-129.
- Hubbard, D. R. and G. M. Berkoff. 1993. Myofascial trigger points show spontaneous needle EMG activity. *Spine* 18(13): 1803-1807.
- Kuhajda, M.C., B. E. Thorn, M. R. Klinger. 1998. The effect of pain on memory for affective words. *Am Behav Med* 20(1): 31-35.
- Joranson D. E., K. M. Ryan, A. M. Gilson. 2000. Trends in medical use and abuse of opioid analgesics. *JAMA* 283(13): 1710-4.

- Llandro, N. I., T. C. Stiles, H. Sletvold. 1997. Memory functioning in patients with primary fibromyalgia and major depression and healthy controls. *J Psychosomatic Res* 42(3):297-306.
- Liebeskind, J. C. 1991. Pain can kill. *Pain* 44(1):3-4.
- Lidbeck, J. 2002. Central hyperexcitability in chronic musculoskeletal pain: A conceptual breakthrough with multiple clinical amplifications. *Pain Res Manag* 7(2):81-92.
- Martinez-Lavin, M. 2001. Overlap of fibromyalgia with other medical conditions. *Curr Pain Headache Rep* 5(4):347-350.
- Meyer, H. P. 2002. Myofascial pain syndrome and its suggested role in the pathogenesis and treatment of fibromyalgia syndrome. *Curr Pain Headache Rep* 6(4):274-83.
- Merskey, H. 1989. Physical and psychological considerations in the classification of fibromyalgia. *J Rheumatol Suppl* 19:72-79.
- Peters A, Schweiger U, Fruhwald-Schultes B et al. 2002. The neuroendocrine control of glucose allocation. *Exp Clin Endocrinol Diabetes* 110(5):199-211.
- Rau, C. L., Russell I. J. 2000. Is fibromyalgia a distinct clinical syndrome? *Curr Rev Pain* 4(4):287-294.
- Simms, R. W. 1998. Fibromyalgia is not a muscle disorder. *Am J Med Sci* 315(6):346-350.
- Simons, D. G. 1999. Diagnostic criteria of myofascial pain caused by trigger points. *J Musculoskeletal pain* 7(1-2):112.
- Simons D. G., J. G. Travell, and L. S. Simons. 1999. *Travell and Simons' Myofascial Pain and Dysfunction: the Trigger Point Manual: Volume I, edition 2: The Upper Body*. Baltimore: Williams and Wilkins
- Starlanyl, D. J. and M. E. Copeland. 2001. *Fibromyalgia & Chronic Myofascial Pain: A Survival Manual*, ed 2. Oakland: New Harbinger Publications.
- Staud, R. C. J. Vierck, R. L. Cannon et al. 2001. Abnormal sensitization and temporal summation of second pain (wind-up) in patients with fibromyalgia syndrome. *Pain* 91(1-2):165-75.
- Travell, J. G. and D. G. Simons. 1992. *Myofascial Pain and Dysfunction: The Trigger Point Manual, Volume II: The Lower Body*. Baltimore: Williams and Wilkins.
- Yaksh, T. L., X. Y. Hua, I. Kalcheva et al. 1999. The spinal biology in humans and animals of pain states generated by persistent small afferent input. *Proc Natl Acad Sci* 96(14):7680-7686.
- Yunus, M. B. 1988. Diagnosis, etiology and management of fibromyalgia syndrome: An update. *Compr Ther* 14(4):8-20.
- Wolfe, F. 1989. Fibromyalgia: the clinical syndrome. *Rheum Dis Clin North Am* 15(1):1-18.