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Fibromyalgia is a complex disorder that involves widespread muscle pain, fatigue, and poor sleep. Patients with fibromyalgia often have a host of other symptoms as well, but the pain, fatigue, and poor sleep dominate the clinical picture with effects on quality of life that range from mild to severe.

Most doctors consider fibromyalgia to be a diagnosis of exclusion. This means that despite a reasonably thorough clinical assessment, no other explanations are found. These other explanations for pain, fatigue, and poor sleep include autoimmune disease, chronic infection, hypothyroidism, anemia, depression, and chronic anxiety.

Descriptions of fibromyalgia-like conditions have been around for centuries. By the mid-20th century most fibromyalgia-like patients were considered psychiatric cases and were treated with drugs.

Every few years, leaders of a psychiatric model for fibromyalgia try to get the band back together but clinical experience shows that a psychiatric interpretation of fibromyalgia is too simplistic. Because the cause of fibromyalgia remains unknown, long-term disability insurers prefer the psychiatric model for reasons of their own.

Fibromyalgia is sometimes lumped together with chronic fatigue syndrome (CFS), which is similar in many ways, but also different than fibromyalgia in important ways. Severe CFS is more properly referred to as ME/CFS, for its similarity to previously described outbreaks of a condition called *myalgic encephalitis*.[1]

The Centers for Disease Control (CDC) have issued a series of diagnostic criteria by which to define a case of ME/CFS for research purposes, and these criteria are notably different from the currently accepted diagnostic criteria for fibromyalgia.[2,3,4]

Diagnosis

The American College of Rheumatology (ACR) proposed the first specific diagnostic criteria in 1990.[5] In addition to excluding other causes of pain, fatigue, and poor sleep,

leaders of the rheumatology model proposed that patients should demonstrate unusual tenderness to light pressure in 11 or more of 18 classic tender point areas on the body.

The ACR criteria helped establish fibromyalgia as a medical condition with psychological implications, as opposed to a primary psychiatric disturbance. Still, a diagnosis of fibromyalgia based on ACR criteria does little to explain what's physically wrong in a given patient with fibromyalgia – or what treatments should be tried.

What's more, the 1990 ACR criteria ignored the many other symptoms commonly reported by patients with fibromyalgia. In addition to pain, fatigue, and poor sleep, people diagnosed with fibromyalgia may also experience a loss of mental clarity, recurrent headaches, chronic sinusitis, irritable bowel syndrome, premenstrual syndrome, and subtle problems involving regulation of the thyroid, adrenal, and autonomic nervous systems.

Neurotransmitter imbalances, while common in fibromyalgia, do not seem to follow a particular pattern. The ACR diagnostic criteria – and the rheumatological paradigm for assessing and treating people with fibromyalgia – therefore lack adequate depth, breadth, and precision.

Recently, a group of experts in the rheumatological model for fibromyalgia revisited the 1990 ACR criteria and proposed a revision.[6] The new system for diagnosing fibromyalgia from this group proposes a dual method of scoring, one for widespread pain, another for symptom severity. The benefit for clinicians using this system is that assessment would no longer require a physical or tender point examination.

The application of these methods might make sense from a research standpoint, but from a clinical standpoint they're clumsy and would interfere with the time consuming process by which integrative clinicians systematically construct a patient's illness narrative.

The clues to the root dynamics of metabolic dysfunction in a given individual with a fibromyalgia are to be found by scrubbing the illness narrative and pursuing additional diagnostic information, not by filling in the blanks of tedious tables for pain locations and symptom severity.

The proposed new criteria also jettison an important physical finding in most people with advanced fibromyalgia known as *allodynia* – tenderness out of proportion to the pressure applied. This tenderness out of proportion can occur in the classic tender point areas, or just about anywhere else.

In a review of the proposed new criteria, Robert Bennett, MD (one of the rheumatologists associated with the 1990 ACR recommendations), cautioned against omitting the "undue sensitivity to touch" criterion, arguing that it would "disparage one seminal feature that is the essence of fibromylagia."[7]

What people with fibromyalgia need are explanations of best fit as to *why* they've developed chronic pain, fatigue, non-restorative sleep, and more, followed by thoughtful care plans aimed at metabolic rehabilitation. Changing the system for documenting pain and symptoms (by making it more complex) will not succeed in turning the diagnosis of

fibromyalgia anything more than a relatively meaningless label for a complex systems biology disorder.

Diseases and syndromes are defined by their pathobiologies – the mechanisms that hinder the functional integrity of the system as a whole. But in most cases of chronic illness, our understanding of the pathobiology of the disease or syndrome in question is limited.

A syndrome like fibromyalgia is much less well defined than a disease like rheumatoid arthritis, but specialty medicine perpetually overestimates the sufficiency of what it knows about almost any given disease. For this reason, many disease definitions amount to professionally rigged constructs completely devoid of inherent explanatory power. [8,9,10]

We advise our patients suspected of having fibromyalgia not to get too hung up on diagnostic labels, just as integrative clinicians avoid getting too hung up on over-documentation of the obvious when it is working explanations, and the results that follow, that count.

Our goal is to understand how each person became ill with their fibromyalgia-like syndrome, and to develop an integrative care plan that is tailored to the uniqueness of that person's health situation. Like all chronic illnesses, fibromyalgia is best viewed as a systems biology puzzle with a systems biology solution.

Pathobiology

Bennett and colleagues recently showed that patients with fibromyalgia have abnormal electroencephalograms (EEGs) compared to healthy controls. Persons with fibromyalgia are more likely to show lower spectral power in the frontal lobes of the brain, especially in the low to mid frequencies.[11] This study also showed that fibromyalgia tend to exhibit a widespread weakness in achieving coherence of brain wave patterns. The more severe their symptoms, the more pronounced these effects became.

Fibromyalgia is generally perceived as a pain amplification syndrome that depends at least in part on mechanical or thermal impulses that originate in the peripheral soft tissues. These impulses travel to spinal cord segments that then process the information and relay it to the brain.

A recent review by Italian researchers acknowledges the evidence for pain amplification in fibromyalgia but draws attention to existing evidence for the multiple abnormalities in the circulation and soft tissues that interact to drive the sensitization of pain receptors in the brain and spinal cord.[12] They state that "a combination of interactions among external stressors, behavioral constructs, neurotransmitters, hormones, immune, and sympathetic nervous appears to be involved."

There is a place in the human body where all of these factors are present: the connective tissue matrix (CTM). Notably missing from the psychiatric and rheumatological models for fibromylagia is any detailed understanding of the role that a dysfunctional CTM plays in the onset and progression of fibromyalgia.

From the clinical systems biology point of view, the most robust model for the treatment of fibromyalgia focuses on *metabolic rehabilitation* – understanding how multiple metabolic systems interact to cause pain and progressive loss of functional integrity, and what can be done to correct it. In this approach, what's going on in the CTM is of paramount importance.

The CTM is also known as the extracellular matrix. It is a mesh-like superstructure containing a mix of collagen and *ground substance*, a mix of proteins, amino acids, and long-chains of sugar molecules known as polysaccharides. When healthy, these ingredients maintain a gel-like state. When unhealthy, the ground substance becomes more tight, brittle, and filled with liquid pockets of inflammation.[13]

Interspersed throughout the CTM super-structure microscopic investigation reveals fibroblasts (the cells that make collagen and elastin fibers (which allow more stretch), and immune cells including lymphocytes, macrophages, and basophils. These cells are all within reach of a rich capillary supply.

The CTM receives thus messages from the hormonal system and the immune system via the blood. Wastes are cleared from the capillaries into the venules which exit the CTM space.

The CTM also contains lymph vessels through which lymphocytes and immune system messenger molecules travel, monitoring local immune system challenges and keeping matrix areas being challenged by noxious stimuli informed about regional and systemic immune system.

All of this activity is being monitored by the autonomic and peripheral nervous systems. The CTM is planted with receptors and terminals from the sympathetic and parasympathetic divisions of the autonomic nervous system, along with pain receptors (nocioceptors), mechanoreceptors, and other types of receptors that feed these sensations into the sensory nerves of the peripheral nervous system. These connect with relay stations in the spinal cord and find a select route to the brain.

The autonomic nervous system signals that arise in the CTM travel up selected pathways to the main sympathetic nerve trunk or to one of a few parasympathetic nerve ganglia (places where nerve can engage in cross-talk and signal processing before sending their message to the brain). These autonomic nerve fibers find their way to the reticular activating system in the brainstem, as well as the hypothalamus and several higher brain centers.

The CTM houses all of these constituents for a reason. Research demonstrates that the CTM contains the elements needed to mount an immediate response to any and all forms of noxious stimulation. The sugar molecules in the CTM include proteoglycans (or PGs, which contain more protein than sugar), glycosaminoglycans (or GAGs, which contain more sugar than protein), and other polysaccharides.

These sugar molecules possess a highly negative charge, which means they can donate electrons to help stabilize noxious stimuli, most of which produce an excess of positively charged protons. Polysaccharides are generally held to be key elements in what may have been the first well-organized adaptive response to noxious stimuli ever evolved by a complex living system.

The neuroimmune system in the CTM carefully monitors changes within the ground substance.Certain changes trigger a tightly integrated adaptive response that involve neurological, circulatory, immune, and endocrine factors.

If the local CTM response fails to adapt or regulate a threat, signals will recruit help in the form of a regionally coordinated response from the spinal cord and autonomic nervous system segments and ganglia connected to the CTM location that is having problems.[14]

If the regional response fails to adapt, the central nervous system gets involved. Suffice it say, if the central, regional, and local responses to noxious stimulation fail to get things under control, you could be on an illness course that will result in some doctor, somewhere, saying, "I think you have fibromyalgia." If common things occur commonly, this insight will earn you a prescription for a fibromyalgia drug.

The drug may help. Yet most of the therapies that have had some success in fibromyalgia produce only mild to moderate benefits, drugs included. There is also a feeling that is common to many patients once they've been informed that they have fibromyalgia, and that's the sense that for the doctor making the diagnosis, thinking has stopped so prescribing could begin.

The various drugs prescribed for patients with a fibromyalgia diagnosis work on the central nervous system or the spinal cord to reduce pain sensations. They do little, if anything, to clear up what's wrong with CTM-level regulatory systems. For this reason, the treatment of fibromyalgia should include an integrative care plan that focuses on clearing peripheral toxicity.

The noxious stimulants capable of overwhelming CTM adaptive responses include mental or emotional stress, repetitive strain, infection (viral, bacterial, fungal, parasitic), antigen invasion (from any protein source including microbes and foods), allergens, chemical toxins, biotoxins (sources include molds, cyanobacteria, dinoflagellates, pfisteria, ciguatera, and "other").

Does it seem like the treatment of fibromyalgia might entail something a little more nuanced than a prescription drug?

Perhaps the most interesting implication of the CTM's involvement in fibromyalgia is that it helps explain why acupuncture, massage therapy, and other forms of bodywork so often help to some degree. These methods stimulate the CTM regulatory responses and may help clear toxicity or low-grade inflammation from these spaces.

Mobilization of toxicity from the CTM into the system might explain why some patients report feeling better right after massage therapy but worse the next day: the CTM cleared somewhat, reducing the flow of peripheral pain impulses, but the system as a whole was not adequately prepared to process the toxic load.

This is why patients with fibromyalgia and undue sensitivity to touch need to be assessed for toxicities and treated with a safe and comprehensive approach to detoxification and the avoidance of noxious stimulation.

One of the hallmarks of fibromyalgia is pain and tenderness involving muscles and soft tissues.

Myofascial Trigger Points

These pains are not examples of garden-variety reactive muscle spasm. Reactive spasm is usually produced as a reaction to injury or to small pockets of inflammation involving the zone where tendon turns into muscle.

The muscle pain of fibromyalgia is associated with multiple so-called myofascial trigger points (MTPs). Reactive spasm demonstrates active muscle fiber contraction sustained by coordinated motor neuron activity. MTPs reveal only spontaneous electrical activity.

One study found that irritating one MTP increased spontaneous electrical activity in a non-irritated MTP in a distant muscle whose nerve input derives from the same spinal segment as the irritated muscle.[15] This study showed that activating pain receptors in one electrically quiet MTP can activate abnormal activity in a distant MTP that had been electrically quiet.

The shortened muscle presents as ropy bands or tender knots under the skin. No specific muscle pathology is seen in biopsies of trigger points, but reports have shown abnormalities in the collagen and fascial tissue within and around affected muscles.[16,17]

Mechanical pressure or chilling of an MTP can excite a sudomotor (sweat) or pilomotor response (goose bumps) that in turn prompts an increase in pain.[18] This indicates the involvement of autonomic nervous system reflexes.

Manual stimulation of MTPs in multiple locations has been shown to completely reproduce the overall fibromyalgia pain pattern experienced by the patient.[19]

Injected local anesthetics are commonly used to reduce MTP pain. In terms of local anesthetic action, procaine is short-acting, bupivicaine is long-acting, and lidocaine's udration of action lies between these two. In terms of trigger point pain relief, the effects of all three are similar.

This family of molecules has been shown to reduce the activity of voltage-gated sodium ion channels in nerve cell membranes.[20] The precise mechanism by which they relieve pain for days to weeks remains unknown.

Comparisons between dry acupuncture needling and lidocaine injection of trigger points have found similar mild to moderate short-term reductions in pain and depression in patients with fibromyalgia.[21] A randomized trial found that lidocaine patches were and lidocaine injection produced similar short-term reductions in trigger point pain.[22]

The overlap in pain characteristics and pathobiology between MTPs and fibromyalgia suggest that fibromyalgia pain is largely drive by MTPs. The question left begging is what causes MTPs to form? The answer is unclear but evidence is mounting for a mechanism involving abnormal function in the connective tissue matrix.

The conventional and integrative approaches to fibromyalgia are best distinguished by the latter's openness to looking beyond mood disorders and central pain amplification mechanisms to other metabolic imbalances that might overload the connective tissue matrix with noxious stimuli that perpetuate the peripheral pain signals that precede the central pain amplification process.

The rheumatological and clinical systems biology models for fibromyalgia place equal emphasis on symptom control, but the integrative approach takes a more systematic and aggressive stance toward uncovering and managing the root dynamics of this debilitating illness.

The conventional medical approach:

- The history and physical exam are often compressed by time constraints but typically include tender point assessment.

- Blood tests and imaging studies are run as needed to rule out autoimmune, infectious, hormone, anemia, or psychiatric causes.

- Treatment emphasizes prescription drugs for pain, sleep, and/or mood. Commonly prescribed drugs include:

    - Savella and Cymbalta are approved for use in fibromyalgia and may help with pain, fatigue, sleep, and/or mood. As of this writing WebMD's *Fibromyalgia Treatment Center* focuses almost exclusively on Cymbalta as treatment for fibromyalgia. At WebMD it thus appears that the highest bidder gets to define the treatment model.

    - Lyrica is approved for neuropathic pain and helps reduce the sensation of pain in some people with fibromyalgia. Lyrica is also prominently featured on consumer health websites pages offering information on fibromyalgia.

    - Pain relievers, from non-steroidal anti-inflammatory drugs (such as ibuprofen) to narcotic analgesics (such as tramadol or hydrocodone), are commonly prescribed to reduce symptoms of pain, with little thought as to underlying issues that cause, aggravate, or amplify pain (such as low serotonin or magnesium levels).

    - Zolpidem, Ambien CR, Lunesta, clonazepam, Xyrem, and other drugs approved for sleep disorders are often used as part of an overall therapeutic strategy in fibromyalgia.

    - Muscle relaxants (such as cyclobenzaprine or carisoprolol) are also used for pain related to spasm – again with little thought given to underlying reasons or perpetuating factors for muscle spasm.

- Note that the conventional treatment of fibromyalgia places little emphasis on lifestyle or natural therapies. While the use of gradual increases in exercise and

of cognitive behavioral therapy are sometimes part of a conventional approach to fibromyalgia, most therapies emphasize can be useful adjuncts to primary therapy, but they are not sufficient as primary therapy for most patients.

If primary care advice doesn't help, referrals are typically made to a rheumatologist and/or psychiatrist, for more of "name the disease, name the drug for the disease" routine, which helps a few, but misses the boat for most. For a description of a systematic rheumatological approach to fibromyalgia, click the link to interview with Daniel J. Clauw, MD, in the reference section.[23]

The integrative approach:

- The history of how the illness unfolded over time is methodically assembled, including multiple sittings if needed. This history generates clues as to how and why health declined gradually over time, or rather suddenly following an infection, accident or other physical trauma, pregnancy, or a period of unrelenting stress. Little research has been done to explore the relationship between environmental exposures and fibromyalgia.

- Physical exam is focused on tender points, blood pressure, nutritional status, muscle tone and reflexes, skin tone, sinuses, throat, lymph nodes, inflammation zones, and any other areas warranted based on the history.

- Treatment emphasizes lifestyle changes and natural therapies and is focused on restoring balance to the metabolic systems that, based on history or exam, appear to have lost their functional integrity. The systems under review typically include those most responsible for maintaining metabolic balance within the system as a whole. They include your systems for:

    - Digestion and assimilation of nutrients (ability to break down and absorb good things while keeping bad things out of the system).

    - Microcirculation of blood and lymph (ability to move oxygen and nutrition in, and trash water out, of tissue zones).

    - Connective tissue matrix regulation (ability of the extracellular matrix and ground substance elements to mount adaptive responses to noxious stimulation).

    - Detoxification (toxin detection, handling, and elimination).

    - Restoration (hormone and neurotransmitter balance).

    - Inspiration (conscious and unconscious brain effects on behavior).

- Exercise and cognitive behavioral therapy are part of a panel of treatment options that include other mind-body techniques for managing stress:

    - Addressing potentially maladaptive psychological defenses.

- Learning techniques for quieting the mind and body at will.
- Meditative practices including contemplative prayer, yoga, and Tai Chi.
- Various stretching and breathing routines.

Therapeutic Lifestyle Change

Eating the healthy foods that are especially good for you, combined with regular exercise, regular periods of relaxation, positive social connections, an optimistic attitude, and a sense of being connected to something greater than yourself together predict not just a longer lifespan, but a longer productive, self-dependent life of quality, dignity, and meaning.

Good quality of life and cognitive behavioral therapy are part of a panel of treatment options that include other mind-body techniques for managing stress:

- addressing potentially maladaptive psychological defenses
- learning techniques for quieting the mind and body at will
- meditative practices including contemplative prayer, yoga, and Tai Chi
- various stretching and breathing routines.

Occasionally a medication regimen will hit a home run for the fibromyalgia patient, but the results of a conventional approach tend to be disappointing. In our experience, Lyrica, Cymbalta, and Xyrem have produced some mild to moderate reductions in pain, fatigue, and non-restorative sleep. Their benefits can show up quickly, too, and that counts for something.

This points out the importance of taking neurotransmitter imbalance into account in patients diagnosed with fibromyalgia, but it should not imply that drugs alone suffice in moderate to severe cases. They don't.

It is not uncommon to see these early benefits wear off after a few months. This loss of therapeutic effect could be due to concurrent problems that remain untreated, such as an irritable bowel syndrome associated with intestinal hyperpermeability and an ongoing ingress of toxins into the system by this route.

The loss of an initial therapeutic drug effect could also mean that the drug mechanism failed due to a developing shortfall in necessary neurotransmitter substrate.

For example, I had a fibromyalgia patient who responded very well to the Cymbalta prescribed by her doctor. After four months, when the effects wore off, increasing the dose did not help. We checked her urinary neurotransmitter levels and discovered that she was not making enough serotonin. The Cymbalta had too little serotonin to work with. We added 5-HTP, the amino acid precursor to serotonin. Her Cymbalta started working again and this correlated with 5-HTP driven increase in her serotonin levels.

Given the studies identifying a broad range of markers of biochemical imbalance in patients with fibromyalgia, I believe the neurotransmitter imbalances that develop in so many of these patients are secondary to deeper, more primary imbalances in their

systems. In other words, a series of case that respond well to a central nervous system drug does not mean that fibromyalgia is a psychiatric condition.

Most patients with fibromyalgia respond slowly or not at all to a narrow-minded conventional approach that emphasizes drug therapy alone. Some who respond early on to medications find that effects wear off or that side effects develop over time

At *onebodymind.com*, we view anxiety the same way we view everything else: as a multidimensional metabolic rehabilitation project that applies specifically to you, and that is amenable to some degree to proper self-care.

Our system of self-assessment and care is consistent with an emerging understanding of the origins of chronic illness that is based on systems biology research. We believe that wise self-care methods should be part of any plan to get key your bodily systems working better together as a team.

Just as the rheumatology model developed more explanatory power than the psychiatric model of fibromyalgia, so will the metabolic rehabilitation model develop more explanatory power than the rheumatology model. This is because the metabolic rehabilitation model is better positioned to develop and apply advances in clinical systems biology, and this will generate more power to explain and treat fibromyalgia.

The challenge for fibromyalgia patients is to find a center with *both* the *experience* needed to apply principles of metabolic balance to complex health problems, and the *time* to apply these principles in a thoughtful and systematic way.

That is how an integrative approach to fibromyalgia looks at the unique big picture for each patient, and continually adjusts itself to achieve better results by restoring functional integrity to metabolic systems that have fallen out of balance.

References:

1. Basset, Jodi. (2004-2009) What is Myalgic Encephalitis?
www.ahummingsbirdsguide.com

2. The 1988 Holmes Definition of Chronic Fatigue Syndrome.
www.cfids-me.org/holmes1988.html

3. The 1994 Fukuda Definition of Chronic Fatigue Syndrome
www.cfids.org/about-cfids/case-definition.asp

4. The 2005 Reeves Definition of Chronic Fatigue Syndrome
www.cdc.gov/CFS/publications/casedef_10.htm

5. Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. American College of Rheumatology 1990 criteria for the classification of fibromyalgia: report of the multicenter criteria committee. *Arthritis Rheum* 1990;33:160-72.
www.rheumatology.org/publications/classification/fibromyalgia/fibro.asp

6. Wolfe F, Clauw DJ, Fitzcharles MA, Goldenberg DL, Katz RS, et al. American College of Rheumatology preliminary diagnostic criteria for fibromyalgia and measurement of symptom severity. *Arthritis Care Research* (Hoboken). May 2010;62(5):600-10. http://www.ncbi.nlm.nih.gov/pubmed/20461783

7. Bennett R. Opinion on preliminary guidelines for the clinical diagnostic criteria of fibromyalgia. *Practical Pain Management*. 2010 Jul/Aug:76-79.

8. Rosenberg, CE. The tyranny of diagnosis: specific entities and individual experience. *Milbank Quarterly*. 2002;80:237-60. http://www.milbank.org/800202.html

9. Slee VN, Slee D, Schmidt HJ. The tyranny of the diagnostic code. *NC Medical Journal*. 2005;66:331-37. http://www.ncmedicaljournal.com/sept-oct-05/Slee.pdf

10. Kibbe DC. Comments and a response to "The Tyranny of the Diagnosis Code" by Slee and colleagues. *NC Medical Journal*. 2005;66:339-40. http://www.ncmedicaljournal.com/sept-oct-05/Kibbe.pdf

11. Hargove JB, Bennett RM, Simons DG, Smith SJ, Nagpal S, Deering DE. Quantitative electroencephalographic abnormalities in fibromyalgia patients. *Clinical EEG and Neuroscience*. 2010 Jul;41(3):132-9. http://www.ncbi.nlm.nih.gov./pubmed/20722346

12. Di Franco M, Iannuccelli C, Valesini G. Neuroendocrine immunology of fibromyalgia. *Annals of the New York Academy of Science*. 2010 Apr;1193(1):84-90. http://www.ncbi.nlm.nih.gov/pubmed/20398012

13. Pischinger A. *The Extracellular Matrix and Ground Regulation: Basis for a Holistic Biological Medcine*. Hartmut Heine (Ed.) 2007. North Atlantic Books: Berkeley, CA.

14. Rea W, Patel K. *Reversibility of Chronic Degenerative Disease and Hypersensitivity. Volume 1*. 2010. CRC Press: Boca Raton, FL.

15. Fernandez-Carnero J, et al. Increased spontaneous electrical activity at a latent myofascial trigger point after nociceptive stimulation of another latent trigger point. *Clinical Journal of Pain*. 2010 Feb;26(2):138-43. http://www.ncbi.nlm.nih.gov/pubmed/20090441

16. Gronemann ST, et al. Collagen and muscle pathology in fibromyalgia patients. *Rheumatology* (Oxford). 2004 Jan;43(1):27-31 http://www.ncbi.nlm.nih.gov/pubmed/12867573

17. Liptan GL. Fascia: a missing link in our understanding of fibromyalgia. *Journal of Bodywork and Movement Therapies*. 2010 Jan;14(1):3-12. http://www.ncbi.nlm.nih.gov/pubmed/20006283

18. Gunn CC. *The Gunn Approach to the Treatment of Chronic Pain*. 2002. Churchill-Livingstone: New York.

19. Prevalence of myofascial trigger points in fibromyalgia: the overlap of two common problems. *Current Pain and Headache Reports*. 2010 Jul 6. [Epub ahead of print]
http://www.ncbi.nlm.nih,gov/pubmed/20607459

20. Vedantham V, Cannon SC. The position of the fast-inactivation gate during lidocaine block of voltage-gated Na+ channels. *Journal of General Physiology*. 1999 Jan;113(1):7-16.
http://www.ncbi.nlm.nih.gov/pubmed/9874784

21. Gazi MC, Issy AM, Avila IP, Sakata RK. Comparison of acupuncture to injection for myofascial trigger point pain. *Pain Practice*. 2010 Jul 29. [Epub ahead of print]
http://www.ncbi.nih.gov/pubmed/20673246

22. Affaitati G, et al. A randomized, controlled study comparing a lidocaine patch, a placebo patch, and anesthetic injection for treatment of trigger points in patients with myofascial pain syndrome: an evaluation of pain and somatic pain thresholds. *Clinical Therapeutics*. 2009 Apr;31(4):705-20.
http://www.ncbi.nlm.nih.gov/pubmed/19446144

23. Clauw DJ. On the effective treatment of fibromyalgia – an update. *ProHealth Interveiw*, September 27, 2007.
http://www.prohealth.com/library/showarticle.cfm?id=3854&t=CFIDS_FM