

Special Article – Fibromyalgia

Fibromyalgia: an Alternative View

McMakin C*

Fibromyalgia and Myofascial Pain Clinic of Portland,
Portland, OR 97214, USA

***Corresponding author:** Carolyn McMakin,
Fibromyalgia and Myofascial Pain Clinic of Portland,
Portland, OR 97214, USA

Received: September 12, 2018; **Accepted:** September 24, 2018; **Published:** October 01, 2018

Short Communication

Fibromyalgia is a complex neuroendocrine pain condition, but it is not incurable. Unfortunately, it cannot be cured simply by managing the symptoms with medication or picking one endocrine item to correct. It is important to discern how the patient came to develop fibromyalgia if you want the patient to actually recover.

As a group, Fibromyalgia patients seem all to have very similar neuroendocrine profiles when they reach the pain clinic. They have symptoms of low thyroid and high but normal TSH. Poor digestion and irritable bowel are common. Abnormal levels of gonadal hormones, low progesterone, testosterone, FSH and LH are characteristic [1-4].

As a group, Fibromyalgia patients have low levels of branch chain amino acids that interfere with proper serotonin and dopamine production and may contribute to abnormal thyroid hormone receptor structure [5]. Pain is amplified in the thalamus instead of being suppressed there [6].

Poor stage-four sleep means low growth hormone. Central suppression of growth hormone releasing hormone (GHRH) by CRF (corticotrophin releasing hormone) compounds this problem making the patient intolerant of even the mildest exercise since growth hormone in an adult mediates repair of muscle tissue after exercise [7,8]. CRF suppresses all central regulatory hormones in fibromyalgia and CRF also serves as a neurotransmitter predisposing the patient to focus on negative or painful events and contributing to what clinicians have come to call “catastrophising”.

All of these neuroendocrine abnormalities occur in a coordinated survival response secondary to physiologic stressors and mediators that cause inflammation and pain. The patient may be unaware of [9] food sensitivities, such as gluten or milk, that can cause inflammation in the gut wall of a susceptible patient and make it permeable to small peptides that can leak across the membrane and activate the immune system that surrounds it. Macrophages ingest these substances and break open to release histamine, which stimulates Class C pain fibers leading to moderate full body pain. Once pain is elevated, it stresses the system and elevates stress hormones that modify central pain processing and central regulating hormones. When the offending food sensitivities are removed, the gut appears to repair itself, the macrophage sensitivity normalizes, and the neuroendocrine system will normalize once the inflammation and pain are gone.

Chronic infection from bacteria, parasites, mold or viruses in the jaw, sinuses, gut or any organ can activate the immune system and cause elevations of cytokines that also increase pain and disturb sleep. Once pain is elevated, it stresses the system and elevates stress hormones that modify central pain processing and the central regulating hormones. In order to reduce the pain, the infection must be managed and alleviated, and the neuroendocrine system will normalize.

Spine trauma that damages a disc annulus can release powerful inflammatory PLA2 capable of stripping the myelin off of the nerves and spinal cord [10-13]. Once the pain pathways in the spinal cord are damaged and conductivity is slowed by PLA2, it creates central or thalamic pain and the patient experiences intense full body pain that includes all the joints and the hands and feet [14]. Fibromyalgia from spine trauma causes elevations of all inflammatory cytokines and substance P [15]. Once pain is elevated, it stresses the system and elevates stress hormones that modify central pain processing and central regulating hormones. In this type of fibromyalgia, pain can be reversed by a topical application of two specific pulsed microcurrent frequencies using 40 hertz and 10 hertz. This treatment reduces pain and all of the cytokines and substance P down to the normal range. If these reduced pain levels are maintained for four months, the neuroendocrine and digestive processes normalize and patients no longer have fibromyalgia.

Fibromyalgia can be reversed, put into remission or cured when the process that caused it can be corrected. This may not be possible in every case, but it should be the intended goal of treatment for every thoughtful, thorough caring physician.

References

1. Crawford LJ, Pillemer SR, Kalogeras KT, et al. “Hypothalamic-Pituitary-Adrenal Axis Perturbations in Patients with Fibromyalgia.” *Arthritis and Rheumatism*. 1994; 37: 1583-1592.
2. Crawford LJ, Engleberg NC and Demitrack MA. “Neurohormonal Perturbations in Fibromyalgia”. *Baillieres Clin Rheumatol*. 1996; 22: 365-378.
3. Crawford LJ. “Neuroendocrine Abnormalities in Fibromyalgia”. *American Journal of the Medical Sciences*. 1998; 315: 359-366.
4. Neek G and Riedel W. “Hormonal Perturbations in Fibromyalgia Syndrome.” *Annals of the New York Academy of Sciences*. 1999; 876: 325-338.
5. Maes M, et al. Serotonergic markers and lowered plasma branched-chain-amino acid concentrations in fibromyalgia. *Psychiatry Research*. 2000; 97: 11-20.
6. Mountz JM, et al. Fibromyalgia in women: abnormalities of regional cerebral blood flow in the thalamus and the caudate nucleus are associated with low pain thresholds levels. *Arthritis Rheum*. 1995; 38: 926-938.
7. Bennett RM. Emerging concepts in the neurobiology of chronic pain: evidence of abnormal sensory processing in Fibromyalgia. *Mayo Clinic Proceedings*. 1999; 74: 385-398.
8. Bennett RM. Adult growth hormone deficiency in patients with fibromyalgia. *Curr Rheumatology*. 2002; 4: 306-312.
9. Sapolsky R. Why zebras don't get ulcers. 1994.

10. Olmarker K, Rydevik B, Nordberg C. Autologous nucleus pulposus induces neurophysiologic and histologic changes in porcine cauda equina nerve roots. *SPINE*. 1993; 18: 1425-1432.
11. Olmarker K, Blomquist J, Stromberg J, Nannmark U, Thomsen P, Rydevik B. Inflammatory properties of nucleus pulposus. *SPINE*. 1995; 20: 665-669.
12. Ozaktay AC, Cavanaugh JM, Blagojev DC. Phospholipase A2 – induced electrophysiologic and histologic changes in rabbit dorsal lumbar spine tissues. *SPINE*. 1995; 20: 2659-2668.
13. Ozaktay AC, Kallakuri S, Cavanaugh JM. Phospholipase A2 sensitivity of the dorsal root and dorsal root ganglion. *SPINE*. 1998; 23: 1297-1306.
14. Kandel E, Schwartz J. *Principles of Neural Science*, second edition. Elsevier Science Publishing Co., Inc., New York. 1985: 331-336.
15. McMakin C, Gregory W, Phillips T. Cytokine changes with microcurrent therapy of fibromyalgia associated with cervical trauma." *Journal of Bodywork and Movement Therapies*. 2005; 9: 169-176.