

*Article 16*

**Etiology, Diagnosis and Treatment of Fibromyalgia:  
A Practical and Effective Approach**

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**Fibromyalgia: A Brief History and Definition**

What we now identify as fibromyalgia syndrome (FMS) has never been an easy condition to diagnose or even describe. Hippocrates, over 400 years before the Common Era, first used the term “rheumatismos” to describe generalized aches and pains. Since that time, FMS has had over 60 named designations.

Until relatively recently, the term most often used to describe generalized, widespread pain was “fibrositis.” This term was proposed in 1904 by Dr. William Gowers in the article “Lumbago: Its Lessons and Analogues.” From 1904 until 1977 there was little organized professional interest in the condition. Then, Smythe and Moldofsky (1977) defined fibrositis in clinical terms by identifying a symptom (widespread pain) and a sign (multiple active tender points). International interest was aroused, and a debate began over the acceptance of “exaggerated tenderness at anatomically reproducible locations” (p. 928) as the definition for diagnosing FMS (Smythe and Molodfsky’s 1977 criteria) versus placing less emphasis on tender points and more focus on the range of associated symptoms for diagnosis (Yunus, Masi, Calabro, Miller, & Feigenbaum, 1981; Yunus, Masi, & Aldag, 1989).

In 1990, amidst debate, the American College of Rheumatology (ACR) developed standardized criteria that became uniformly accepted by the American medical community (Wolfe et al., 1990). ACR criteria define FMS as a musculoskeletal condition that is manifest when a person has a history of chronic, diffuse pain that is characterized by the finding of bilateral tender points at 11 out of 18 sites when measured by pressure applied at these sites.

Whatever the criteria set for judging tender points, other associated patient complaints include sleep disturbance, fatigue, morning stiffness, swelling, pain that intensifies with menses, pain that intensifies with changes in the weather, anxiety, depression, confusion, memory loss, and numerous other somatic and psychological symptoms. The fatigue, weakness, and numerous non-rheumatic symptoms and syndromes which occur in individuals who are diagnosed with FMS have caused some medical researchers to postulate that FMS has its origin in either immunologic dysfunction (e.g., Caro, 1989) or central nervous system (CNS) dysregulation (Clauw, 1995a, 1995b). Because of the high level of somatic complaints and concomitant anxiety and depression, other researchers (primarily psychologists and psychiatrists) have viewed FMS as a somatic disorder (e.g., Hudson & Pope, 1989; Krag, Norregaard, Larsen, & Danneskiold-Samsoe, 1994).

Clinical diagnosis of fibromyalgia has been based on the exclusion of other illnesses for which objective criteria are found. This puts FMS in the category of “symptom-based” conditions that are “characterized by chronic physical (somatic) symptoms” (Hyams, 1998, p. 148). Symptom-based conditions have certain shared general features, making them difficult to differentiate from one another.

Setting diagnostic criteria for somatic conditions has been especially difficult. Hyams (1998) stated the problem as one of specificity, reflecting that diagnostic criteria have not been developed that can consistently exclude acknowledged medical and psychiatric causes of FMS-like chronic physical conditions. Correct diagnosis of these diseases, and others without a pathognomonic sign, have initially relied upon distinctive clinical signs. When diagnostic

criteria of specific physical or biological abnormalities are established, diagnosis can be made based on assessment of the relevant biological factors.

Recent research has provided clues to the physiological processes that inform the pain mechanism in fibromyalgia. With the identification of these processes, more exact diagnosis and treatment can be implemented. This paper summarizes these recent findings and proposes a “Best Practices” treatment protocol that features cognitive-behavioral therapy.

**Psychological Findings of Abuse and Stress are  
Relevant to the Development of the Chronic Pain  
Experienced by Individuals with FMS**

Essentially, what has become clear through recent research, (e.g., Liu, Mantyh, & Basbaum, 1997), is that persistent physical or emotional abuse, experienced as chronic stress by a person, may cause certain physiological changes in the individual. There is a significant group of individuals diagnosed with FMS who experienced chronic stress as a result of both physical and emotional abuse, and who have certain common (and specific) physiological findings. These findings include elevated levels of the neurotransmitter N-methyl-D-aspartate-substance P (NMDA-SP) and an increase in the expression of *c-fos* (a protein produced under stress and found neurologically present when pain is reported). These clinical findings are robust and point to chronic stress as the key to the development of the hyperalgesia of FMS. The resultant hyperalgesic state (FMS) is a condition of chronic muscular oversensitivity to both pain and fatigue, better identified as a stress-induced, diathetic, neuromuscular hyperalgesic condition.

Of the FMS researchers, Bennett (1999), alone, addressed the concept of chronic stress influencing the development of FMS. He concluded that once a complete physiological understanding of the development of chronic nonnociceptive pain is achieved, then conditions such as FMS will not be considered as somatoform disorders. Bennett went even further by asserting that there may be

a “reappraisal” of the concept of somatoform disorders, designating them as medical conditions because the physiology is understood.

Bennett (1999) describes the physiological pain pathway as a “cascade of impulses that originates from nociceptors in somatic or visceral tissues” (p. 386). In the pathway, nerve “impulses travel in peripheral nerves, with a first synapse in the dorsal horn and a second synapse in the thalamus, and end up in the cerebral cortex and other supraspinal structures. This results in a pain experience and the activation of reflex and later reflective behaviors” (p. 386).

Usually nociceptor-driven pain is successfully stopped. With chronic pain, however, the relationship between nociception and recovery from pain is inappropriate or even absent and, therefore, pain continues. Normally the pain threshold *increases* with palpation, but in FMS, the pain threshold *decreases* (Kosek, Ekholm, & Hansson, 1996). It appears this may be caused by absent or inadequate recovery of nociceptor impulses.

I would argue that understanding the physiology of how stress translates into chronic pain illuminates how mind and body interact, rather than simply describes a medical process. The growth of knowledge regarding chronic stress and its relationship to nonnociceptive pain will lead to the recognition of how psychological and social conditions inform physical pathways. Physiological research into the cellular mechanisms of the stress-chronic pain connection argues for a unified mind-body theory of hyperalgesic pain: the same syndrome commonly known as fibromyalgia.

### **Psychological Assessment as Part of the Diagnostic Protocol for Identifying FMS**

A major theme of this paper has been the discussion of the effects of stress upon chronic pain pathways. Psychological research has identified a history of early abuse and high stress levels in FMS individuals (e.g., Boisset-Pioro, Esdaile, & Fitzcharles, 1995; Goldberg, Pachas, & Keith, 1999; McBeth, MacFarland, Benjamin, Morris, & Silman, 1999). The literature is also clear that “virtually all

studies have demonstrated a greater degree of lifetime or daily stresses among patients with FMS as compared with normal controls” (Baldry, 2001, p. 367).

Even when other psychological states are discussed in relationship to FMS, stress has been identified as an “important confounding variable for other psychological distresses including anxiety or depression” in FMS (Dailey, Bishop, Russell, & Fletcher, 1990; Uveges et al., 1990). Because of this common acknowledgment, I believe that it is reasonable to say that there is a putative stress-FMS connection.

This connection not only supports the theory discussed in this paper, it argues that psychological screening be part of the diagnostic process of all individuals suspected of having FMS. As with the suggested medical tests, the addition of a psychological assessment component has implications for treatment.

Although there are widely accepted, reliable, and valid assessments that could be chosen to evaluate stress and anxiety in the individual (e.g., PAI and MMPI), for initial screening I propose that the State-Trait Anxiety Inventory (STAI), developed by Charles Spielberger, Ph.D., be given primary consideration (Spielberger, 1985).

There are several reasons that I suggest the STAI. First, research (i.e., Gaudry, Vagg, & Spielberger, 1975; Spielberger, 1985) has shown that this assessment allows the examiner to identify both temporary, or transient, conditions of “state anxiety” usually associated with feelings of nervousness which increase in response to psychological stress and the longer-standing “trait anxiety” associated with a history of chronic stress. Since either (or both) types of stress have been identified in virtually all individuals with FMS, the STAI would allow the practitioner to ascertain the existence, and type(s), of stress as part of the initial examination. Second, the STAI is normed for high school to adult populations, the most common populations where FMS is diagnosed. Third, the STAI has a brief administration time of only 10 minutes, and can be hand-scored in a few minutes by an assistant.

## **Proposed Diagnostic Protocol for Identification of FMS and Potential Concomitant Problems**

Currently, the only observations needed to diagnose FMS are (a) a history of widespread pain, present for at least 3 months and (b) the identification of pain in 11 of 18 tender points on palpation.

As discussed, there are certain neuroendocrine and psychological factors that have now been identified as occurring in virtually all individuals who are diagnosed with FMS. I propose that adding the following diagnostic elements to the existing protocol (above) would result in a more comprehensive and accurate identification of FMS.

1. Assessment of long-standing stress as measured on a scale of state-trait anxiety.
2. Assessment of the various physiological findings common in individuals with FMS. These include, but may not be limited to the following:
  - a) Assessment for blunted TSH response and the lowered free serum calcium and calcitonin
  - b) Assessment for altered hypothalamic-pituitary-adrenal axis (H-P-A) due to exaggerated adrenocorticotrophic hormone response to corticotropin-releasing hormone (CRH) and a subsequent reduced secretion of cortisol.
  - c) Assessment for elevated levels of Substance P

### **A Comprehensive, Research-Based, Treatment Model for FMS**

The information and deductions presented in this paper inform a certain treatment protocol that differs from the approaches currently in use and summarized earlier in this paper. The primary element of the proposed treatment protocol are (a) the use of cognitive psychotherapy with all FMS patients; and (b) the addition of psychological assessment to the diagnostic and treatment protocols.

## **Psychotherapy as a Primary Component in the FMS Treatment Protocol**

If stress-induced, neuromuscular reactivity resulting in chronic pain is the primary mechanism in the development of FMS, it follows that treating the stress condition is elemental to the recovery process. As mentioned above, all of the medical researchers acknowledge, to one extent or another, that psychotherapy is useful in treating FMS, but none make psychotherapy a requisite treatment in their protocols. Psychotherapy is not just useful, it is as essential to the FMS treatment protocol as are the medical interventions. This is because, without psychological treatment, the conditions that induced the stress will still be present and will therefore continue to cause the physiological dysregulation to occur, in spite of any medical interventions.

Of all the psychological therapies, CBT has been reported as having consistent efficacy in reducing the symptoms of FMS (Bradley, 1989; Nielson, Walker, & McCain, 1992; Goldenberg, et al., 1994; Singh, Berman, Hadhazy, & Creamer, 1998; White & Neilson, 1995). Most psychological studies with FMS patients have included relaxation training, pain and body education, reinforcement of healthy behavior patterns, coping skills training, pain management training, and support in helping the patient restructure maladaptive beliefs about the ability to control pain under the umbrella of CBT techniques.

Of special interest are the only long-term review studies of the use of CBT with FMS patients. These studies (Singh et al., 1998; White & Nielson, 1995) found significant long-term improvement (up to 30 months post program) in FMS symptoms, using the CBT model. These findings, in particular, support the concept that mediating stress effectively decreases the symptoms of FMS, and suggests that cognitive behavioral psychotherapy is essential in the treatment of FMS.

Why CBT interventions have been demonstrated to be the most effective with FMS is that, of the different psychological

approaches, CBT has been shown to lower the stress reaction by helping the client increase their adaptive strategies through understanding the specific causes of the stress. Beck (1967) in his landmark book, *Depression: Causes and Treatment*, pointed out that patients, through the use of cognitive techniques, are able to change maladaptive attitudes that have resulted from early experiences and sensitization to “particular types of stresses” (p. 319). This adaptation allows the individual to gain mastery of their problems, thus reducing chronic stresses and the resulting pain responses to chronic stress. It is this reduction of the chronic stress reaction that is elemental to managing the stress-induced pain response of FMS.

### **Conclusion**

Historically, much confusion has surrounded the identification and treatment of fibromyalgia. Although the condition was identified hundreds of years ago, consensus by the medical community as to common diagnostic features only occurred several years ago. To date, there has not been an agreement as to the etiology of the condition and no single comprehensive protocol has been established for its treatment.

Numerous research efforts, resulting in hundreds of articles, have been undertaken in order to determine the *medical* causes of Fibromyalgia Syndrome. Not unlike the parable of the blind men describing the elephant and trying to describe what an elephant looks like, these research efforts have resulted in describing various attributes of FMS, but not the cause of the condition. Hormonal alterations, painful body locations, endocrine changes, and the other physical findings of FMS are extrinsic to causation.

Certainly, for purposes of treating co-occurring medical problems, it is important to be aware that these conditions could exist in the patient with FMS. However, treating the symptoms of a condition is not the same as treating the cause, or better yet, preventing the condition.

This paper offers both a research-based explanation of why

myofascial pain occurs with FMS, and proposes a comprehensive treatment approach that is grounded in current investigations.

If this premise is borne out, then certainly better treatments can be developed, and, for the first time in the hundreds of years of documentation of this condition, prevention can be considered.

## References

- Baldry, P. E. (2001). *Myofascial pain and fibromyalgia syndromes: A clinical guide to diagnosis and management*. New York: Churchill-Livingstone.
- Beck, A. (1967). *Depression: Causes and treatment*. Philadelphia: University of Pennsylvania Press.
- Bennett, R. M. (1999). Emerging concepts in the neurobiology of chronic pain: Evidence of abnormal sensory processing in fibromyalgia. *Mayo Clinical Procedures*, 74, 385-398.
- Boisset-Pioro, M. H., Esdaile, J. M., & Fitzcharles, M. A. (1995). Sexual and physical abuse in women with fibromyalgia syndrome. *Arthritis and Rheumatism*, 38(2), 235-241.
- Bradley, L. A. (1989). Cognitive-behavioral therapy for primary fibromyalgia. *Journal of Rheumatology*, 19(Suppl), 131-136.
- Caro, X. J. (1989). Is there an immunologic component to the fibrositis syndrome? *Rheumatological Disease Clinics of North America*, 15, 169-186.
- Clauw, D. J. (1995a). Fibromyalgia: More than just a musculoskeletal disease. *American Family Physician*, 52(3), 843-850.
- Clauw, D. J. (1995b). The pathogenesis of chronic pain and fatigue syndromes, with special reference to fibromyalgia. *Medical Hypotheses*, 44, 369-378.
- Dailey, P. A., Bishop, G. D., Russell, I. J., & Fletcher, E. M. (1990). Psychological stress and the fibrositis / fibromyalgia syndrome. *Journal of Rheumatology*, 17(10), 1380-1385.
- Gaudry, E., Vagg, P., & Spielberger, C. (1975). Validation of the state-trait distinction in anxiety research. *Multivariate Behavioral Research*, 10(3), 331-341.

- Goldberg, R. T., Pachas, W. N., & Keith, D. (1999). Relationship between traumatic events in childhood and chronic pain. *Journal of Disability and Rehabilitation, 21*(1), 23-30.
- Goldenberg, D. L., Kaplan, K. H., Nadeau, M. G., Brodeur, C., Smith, S., & Schmid, C. H. (1994). A controlled study of a stress-reduction, cognitive behavioral treatment program in fibromyalgia. *Journal of Musculoskeletal Pain, 2*, 53-66.
- Gowers, W. R. (1904). Lumbago: Its lessons and analogues. *The British Medical Journal, 1*, 117-121.
- Hudson, J. I., & Pope, H. G. (1989). Fibromyalgia and psychotherapy: Is fibromyalgia a form of 'affective spectrum disorder'? *Journal of Rheumatology Supplement, 19*, 15-22.
- Hyams, K. C. (1998). Developing case definitions for symptom-based conditions: The problem of specificity. *Epidemiologic Reviews, 20*(2), 148-156.
- Kosek, E., Ekholm, J., & Hansson, P. (1996). Sensory dysfunction in fibromyalgia patients with implications for pathogenic mechanisms. *Pain, 68*(2-3), 375-383.
- Krag, N. J., Norregaard, J., Larsen, J. K., & Danneskiold-Samsoe, B. (1994). A blinded, controlled evaluation of anxiety and depressive symptoms in patients with fibromyalgia, as measured by standardized psychometric interview scales. *ACTA Psychiatrica Scandinavica, 89*, 370-375.
- Liu, H., Mantyh, P. W., Basbaum, A. I. (1997). NMDA-receptor regulation of substance P release from primary afferent nociceptors. *Nature, 386*, 721-724.
- McBeth, J., MacFarland, G. J., Benjamin, S., Morris, S., & Silman, A. J. (1999). The association between tender points, psychological distress, and adverse childhood experiences: A community-based study. *Arthritis and Rheumatism, 42*(7), 1397-1404.
- Nielson, W. R., Walker, C., McCain, G. A. (1992). Cognitive behavioral treatment of fibromyalgia syndrome: preliminary findings. *Journal of Rheumatology, 19*(1), 98-103.

- Singh, B. B., Berman, B. M., Hadhazy, V. A., & Creamer, P. (1998). A pilot study of cognitive behavioral therapy in fibromyalgia. *Alternative Therapies in Health and Medicine*, 4(2), 67-70.
- Smythe, H. A., & Moldofsky, H. (1977). Two contributions to understanding of 'Fibrositis' syndrome. *Bulletin of Rheumatological Diseases*, 28, 928-931.
- Spielberger, C. (1985). Assessment of state and trait anxiety: Conceptual and methodological issues. *Southern Psychologist*, 2(4), 6-16.
- Uveges, J. M., Parker, J. C., Smarr, K. L., McGowan, J. F., Lyon, M. G., Irvin, W. S., et al. (1990). Psychological symptoms in primary fibromyalgia syndrome: Relationship to pain, life stress, and sleep disturbance. *Arthritis and Rheumatology* 33(8), 1279-1283.
- White, K. P., & Neilson, W. R. (1995). Cognitive behavioral treatment of fibromyalgia syndrome: A follow-up assessment. *Journal of Rheumatology*, 22(4), 717-721.
- Wolfe, F., Smythe, H. A., Yunus, M. B., Bennett, R. M., Bombardier, C., & Goldenberg, D. L., et al. (1990). American College of Rheumatology 1990 Criteria for the classification of fibromyalgia. (Report of the Multicenter Criteria Committee). *Arthritis and Rheumatism*, 33, 160-172.
- Yunus, M. B., Masi, A. T., & Aldag, J. C. (1989). Preliminary criteria for primary fibromyalgia syndrome (PFS): Multivariate analysis of consecutive series of PFS, other pain patients and normal subjects. *Clinical Experimental Rheumatology*, 7, 63-69.
- Yunus, M. B., Masi, A. T., Calabro, J. J., Miller, M. A., & Feigenbaum, S. L. (1981). Primary fibromyalgia (fibrositis): Clinical study of 50 patients with matched normal controls. *Seminars in Arthritis and Rheumatism*, 11, 151-171.

