Fibromyalgia: Is It Real?

This introductory article is the first of a series of three papers on fibromyalgia. The subsequent two papers will focus on evidence-based treatments and newer emerging therapies to improve function and hope.

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Fibromyalgia syndrome (FMS), has generated considerable controversy in the past 20 years. The hallmark characteristic is chronic widespread pain. This is often accompanied by fatigue, non-restorative sleep, mood disorders and somatic symptoms. The challenges are multiple with an unknown pathogenesis, variable symptoms, unpredictable treatment responses and high prevalence rates (reported to be 2-3% in Canada, with females nine times more commonly affected than males).[1] The lack of an objective diagnostic laboratory test and of a gold standard of treatment further complicates management. This has resulted in physician insecurity in managing such patients. Medical school education regarding pain mechanisms and management is limited and extends to the postgraduate level as well.[2] This results in FMS being a frequently neglected, poorly understood and treated condition.

Scientific Basis for Fibromyalgia

Recent neurophysiologic studies including functional MRI (studying dynamic brain activity in response to pain stimuli) have provided validation of reported widespread pain in the absence of an identifiable physical abnormality (Figures 1 and 2).[3-6] These findings are currently available in the research setting only, and not yet available for general community use in diagnosing and following FMS patients.

Abnormalities in pain processing have been identified at various levels in the peripheral, central, and sympathetic nervous systems, as well as the hypothalamic-pituitary-adrenal (HPA) axis stress-response system.[3-8] Documented abnormalities include evidence of peripheral sensitization (pressure and thermal hyperalgesia), central sensitization (dynamic mechanical allodynia, and pinprick hyperalgesia with wind-up phenomenon and after-sensation), increased levels of substance P, glutamate and nerve growth factor in

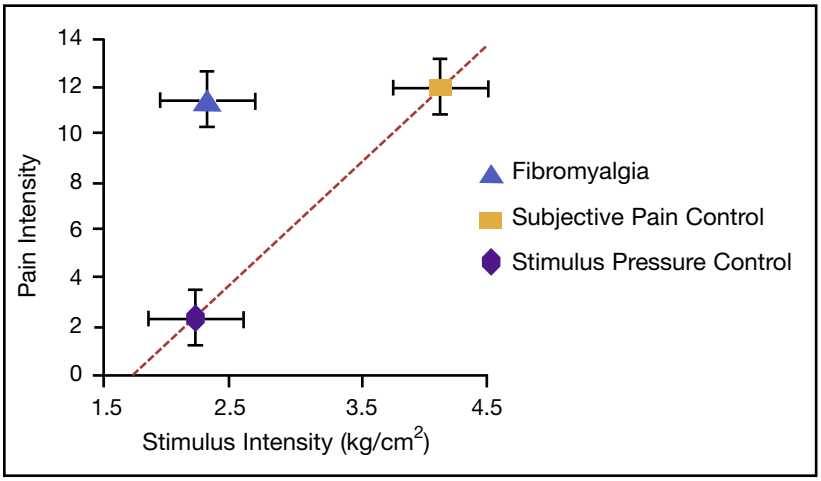

Figure 1: Pain Intensity vs. Stimulus Intensity[3]

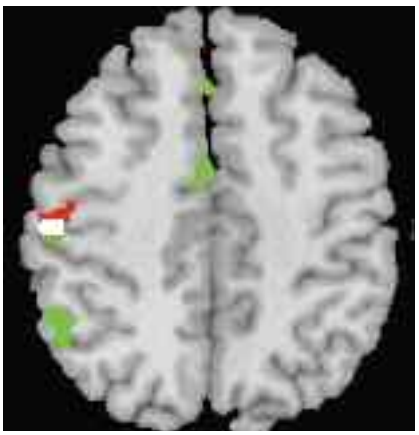

Figure 2: fMRI: Enhanced responses in somatosensory primary and secondary cortex, insula, putamen, and cerebellum[3]

Table 1
1990 ACR Classification Criteria[13]

History of widespread pain > three months
- Above and below waist
- Left and right side
- Must include axial spine

Pain in 11 of 18 tender points, palpation pressure < 4 kg
- Suboccipital muscle insertions
- Low cervical (C5 – C6 transverse processes)
- Upper middle trapezius muscles
- Supraspinatus origins (medial upper scapula)
- 2nd ribs (near costochondral junction)
- Lateral epicondyles (2 cm distal to them)
- Gluteal muscles (upper outer quadrant)
- Greater trochanters (2 cm posterior)
- Medial fat pad of knees (above joint line)

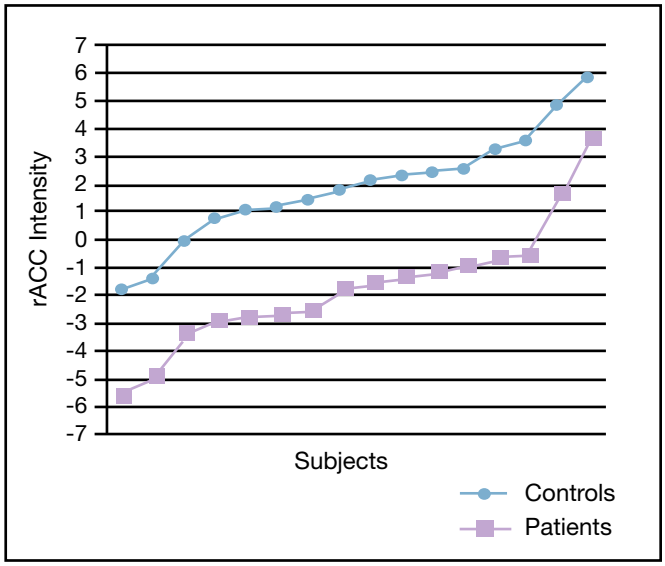

Figure 3: Intensity of Activation in rACC (the Origin of DNIC) During Provoked Pain[4]

the cerebrospinal fluid, and loss of descending noxious inhibitory control, or DNIC, (Figure 3). Recent research also points to interactions between peripheral, central bulbo-spinal and central cortical mechanisms with documentation on functional MRI and SPECT scan imaging of the CNS.[6,7]

Genetic factors may predispose some individuals to a dysfunctional stress response via the HPA-axis.[8] The odds risk ratio is 8.5 for first degree relatives.[9] In addition, there is epidemiological evidence that early life adversity such as the death of a mother, being in institutional care or family financial hardships are linked to chronic widespread pain in adult life.[10] These numerous interacting sensitizing factors may be the setting in which an inciting event such as a viral illness, accident (the odds are 13 times greater after a motor vehicle accident neck injury vs. a leg fracture),[11] or emotional trauma (reported in 31% of patients),[12] then becomes the trigger for FMS in some patients.

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Diagnosing FMS in Clinical Practice

FMS should be positively diagnosed and no longer be seen as a diagnosis of exclusion. The 1990 American College of Rheumatology (ACR) diagnostic criteria were developed for research purposes and not purely as a diagnostic instrument (Table 1).[13] This older criteria require the presence of widespread pain for at least three months duration with the presence of at least 11 of 18 tender points in designated areas (Table 1). Tender points, the only physical examination finding used to help diagnose FM, indicate an overall reduction in pain threshold. They do not represent localized soft tissue pathology, and have been criticized for reliance on subjective interpretation. New ACR criteria are currently being reviewed to eliminate overreliance on tender points in favour of incorporating other clinically meaningful symptoms (sleep disturbance, fatigue, cognitive impairment) and somatic symptoms (depression, headache, irritable bowel) frequently present in FMS patients.[14] An about to be validated tool that the author has found to be very helpful in screening for FMS is the Fibromyalgia Moldofsky Questionnaire. A score > 11 is suggestive of FMS.

As there is no single routine lab test that confirms a diagnosis of FMS, it is recommended that only minimal testing, including a complete blood count (CBC),

Table 2
Modified Diagnostic Criteria 2010[14]

Patient must score 13 or more when the two categories are summed (with a minimum of 3 on the WPI):

Widespread Pain Index	Symptom Severity Scale
Presence of pain in any of the following 19 locations: • Jaw (right and left) • Shoulders (right and left) • Upper arm (right and left) • Lower arm (right and left) • Hips (right and left) • Upper leg (right and left) • Lower leg (right and left) • Neck • Chest • Abdomen • Upper back • Lower back ___/19 points	For each symptom, indicate severity over past week: 0 = no problem 1 = slight problems; generally mild or intermittant 2 = moderate, considerable problems 3 = severe, continuous, pervasive, life-disturbing ___ Fatigue ___ Waking unrefreshed ___ Cognitive symptoms ___ Somatic symptoms* ___/12 points *Somatic symptoms include: depression, headache, and abdominal pain

erythrocyte sedimentation rate (ESR), thyroid function tests (TSH), and creatinine kinase (CK), is done in the first instance. Other investigations (for rheumatic diseases or neurological disorders), or referral to a specialist should be driven by abnormal clinical findings.

Excessive and unnecessary testing may be detrimental to the well-being of patients by promoting an illness-centered focus and fostering a sense of uncertainty. Useful testing includes sleep studies, which should be done and interpreted by neuropsychiatry specialists with an understanding of the non-restorative sleep architecture disturbances seen in FMS (and not for simply ruling out sleep apnea). Quantitative Sensory Testing (QST, available at the CCIM drkoprp.com) goes beyond the traditional EMG-nerve conduction studies in being able to assess the slower pain conducting A-delta and C nerve fibres.

While a positive diagnosis of FM may alleviate patient concerns and is associated with reduced healthcare utilization and decreased investigations, many continue to question the validity of reported functional disability. A positive diagnosis also has medico-legal and work ability implications.[15] Dx

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Take-Home Message

- FMS is a valid medical condition with pathophysiological functional abnormalities in the CNS (including functional MRI and neurophysiological studies demonstrating central sensitization and loss of DNIC)
- The poor understanding of the pathogenesis of FMS clinically leads to doubts about the validity of FMS complaints and results in physician insecurity and reluctance in caring for such patients
- FMS is not a diagnosis of exclusion; a positive diagnosis of FMS should be made without extensive and unnecessary investigations. A useful screening tool is the FMS Moldofsky Questionnaire
- The 1990 diagnostic criteria was based on widespread pain and tender points; the newer 2010 criteria will incorporate associated symptoms of non-restorative sleep, fatigue, and cognitive impairment as well as somatic symptoms (*e.g.*, headache, abdominal pain and depression)
- Minimal testing should be done in the first instance; other investigations should be driven by abnormal findings on clinical history and examination
- QST is an emerging assessment tool for identifying the pattern and distribution of pain
- Education of the patient, open discussion about his/her expectations ("What worries you the most about your symptoms?"), and identification of root causes ("What happened at the very onset of symptoms?") with subsequent management plan are required to provide validation and hope to patients

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