Fibromyalgia: Challenges in Diagnosis, Classification, and Management

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Fibromyalgia Key Points

• Represents the end of the spectrum of chronic musculoskeletal pain. It is not a disease.
• Characterized by chronic widespread pain, insomnia, fatigue, distress, and autonomic symptoms.
• Patients display extensive cutaneous hyperalgesia (allodynia), but most of their pain complaints are related to muscles, joints and ligaments.
• Pain-processing abnormalities- central sensitization (amplification).
• Effective treatments include cognitive behavioral therapy, aerobic exercise, and pharmacologic rx.
There are over 100 named musculoskeletal disorders.

Pain is the most common musculoskeletal symptom.
PATHOPHYSIOLOGY OF PAIN

1800'S

2016

2050?
Pain:

• Opposite of pleasure - philosophical
• Punishment for wrongdoing - moral
• Suffering - psychological
• Warning system to protect the body - pathophysiologic
Acute Pain:

Associated with acute disease or traumatic injury and subsides as healing occurs.

e.g. low back pain, gout
Chronic Pain

- Persisting after healing is complete.
- Associated with an ongoing disease process (osteoarthritis, RA).
- Begins with no identifiable precipitating event or organic cause (fibromyalgia).
Complexities of Pain

• Reliance on the patient’s perception and description
• Individual variability of pain
• Role of psychological and social factors
Pain Spectrum

Localized

Acute

Widespread

Chronic

Time

Localized:

Acute

Widespread:

Chronic

PAIN COMES AND GOES WITHIN 3 MONTHS

0 No Hurt
2 Hurt Little Bit
4 Hurt More
6 Hurt Even More
8 Hurt Whole Lot
10 Hurts Worst
Fibromyalgia Versus Chronic Widespread Pain

- Chronic widespread pain (CWP) occurs in 11%-14% of the population\(^1,2\)
  - Prevalence increases with age to 23% among those in their sixties\(^3\)

- FM is a CWP condition that is also characterized by tenderness, disturbed sleep, and pronounced fatigue\(^4,5\)
  - Patients with CWP have <11 of 18 tender points
  - FM is less common than CWP and is part of a continuous spectrum of chronic pain\(^3\)

Fibromyalgia (FM) is a chronic pain condition and is distinct from other types of pain.

Pain is the most common reason for physician visits.

- **Nociceptive Pain**
  - (eg, burns, cuts)
  - Painful Stimuli

- **Neuropathic Pain**
  - (eg, herpes zoster, DPN)
  - Neuronal Damage

- **Inflammatory Pain**
  - (eg, rheumatoid arthritis, psoriatic arthritis)
  - Inflammation

- **Central Pain Amplification**
  - (eg, FM)
  - Abnormal Pain Processing by CNS
Fibromyalgia:

1. **Widespread** musculoskeletal pain present for at least 3 months.

2. Pain in 11 of 18 **tender point** sites with digital palpation.
## Historical Perspective on Fibromyalgia

<table>
<thead>
<tr>
<th>Muscular rheumatism</th>
<th>Fibrositis</th>
<th>Fibromyalgia</th>
<th>Central amplification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1700-1850</td>
<td>1904</td>
<td>1990</td>
<td>2002</td>
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</tbody>
</table>
“It is the balance of evidence that determines diagnosis.”

“Patients assume, quite rightly, that all symptoms have a cause and also, often wrongly, that obtrusive symptoms have an obtrusive cause.”

“Always scrutinize carefully alleged causes.”
THE AMERICAN COLLEGE OF RHEUMATOLOGY 1990 CRITERIA FOR THE CLASSIFICATION OF FIBROMYALGIA

Report of the Multicenter Criteria Committee

FREDERICK WOLFE, HUGH A. SMYTHE, MUHAMMAD B. YUNUS, ROBERT M. BENNETT, CLAIRE BOMBARDIER, DON L. GOLDENBERG, PETER TUGWELL, STEPHEN M. CAMPBELL, MICHA ABELES, PATRICIA CLARK, ADEL G. FAM, STEPHEN J. FARBER, JUSTUS J. FIECHTNER, C. MICHAEL FRANKLIN, ROBERT A. GATTER, DANIEL HAMATY, JAMES LESSARD, ALAN S. LICHTBROUN, ALFONSE T. MASI, GLENN A. McCAIN, W. JOHN REYNOLDS, THOMAS J. ROMANO, I. JON RUSSELL, and ROBERT P. SHEON

Arthritis and Rheumatism 33:1990
FM Is Characterized by Chronic Widespread Pain and Tenderness

- American College of Rheumatology (ACR) criteria for the classification of FM include:
  - Chronic widespread pain (core feature) for ≥3 months
  - Pain above and below the waist
  - Pain on left and right sides of body
  - Pain in the axial skeleton
  - Pain at ≥11 of 18 tender points when palpated with 4 kg/cm$^2$ of digital pressure

- Other proposed FM criteria, intended to complement ACR classification criteria, have been developed.


The ACR criteria are:
- Sensitive (84.7%) proportion of patients correctly identified as having the condition
- Specific (81.1%) proportion of patients correctly identified as not having the condition
**FM Epidemiology and Risk Factors**

- Prevalence of FM in United States is estimated to be 2% to 5% of the adult population
- FM is often underdiagnosed/misdiagnosed
  - Diagnosis takes an average of 5 years
- Most common in individuals aged 25 to 60 years
- Risk factors include:
  - Genetic: increased incidence among first-degree relatives, associated with genetic markers
  - Environmental: physical trauma, infections, social stressors
  - Gender: more common in women- 70-80%
Tender Points

• Definition
  – Location- Why these sites?
  – Terminology- trigger points, tender points, myofascial, complex regional pain syndromes

• Factors involved
  – Structural- muscle, ligament, tendon, nerve
  – Local vs. referred- pain generators, mechanical
  – Genetic
  – Disease states- RA, OA, SLE, spinal disorders, emotional
  – Central pain processing mechanisms
Tender Points

- Forearm muscles attach to lateral epicondyle
- Gluteus maximus
- Gluteus medius
- Iliotibial tract
- Trochanteric Bursitis
MANUAL TENDER POINT SURVEY

Score on a scale of 0 to 10.

0 = No Pain

10 = The worst pain that you have ever experienced
Patient Position:
- Seated, head in neutral position

Examiner Position:
- Beside

Procedure:
1. Identify the tip of the mastoid process and cricoid cartilage (C6) below the thyroid cartilage
2. Move the thumb straight down from the mastoid process to C5-C7 range (cricoid level)
3. Support the other side of the neck
4. Press toward the opposite shoulder
Pain sensitivity in the general population

- As with most other physiological processes, the processing of pain and other sensory information by the brain and spinal cord is governed by a “volume control setting”
- Volume control is likely set genetically and modified by environmental influences
- The higher the volume control setting, the more pain experienced, irrespective of peripheral nociceptive input
Tender-Point Pain:
Fibromyalgia Patients versus Controls

Patient Self-report Survey for the Assessment of Fibromyalgia Based on Criteria in the 2011 Modification of the ACR Preliminary Diagnostic Criteria for Fibromyalgia

The possible score ranges from 0 to 31 points; a score ≥13 points is consistent with a diagnosis of fibromyalgia.
The “Systemic” Conditions That Overlap With Fibromyalgia

- Tension/migraine headache
- Affective disorders
- Temporomandibular joint syndrome
- Memory and cognitive difficulties
- ENT complaints (sicca syndrome, vasomotor rhinitis, accommodation problems)
- Vestibular complaints
- Multiple chemical sensitivity, “allergic” symptoms
- Esophageal dysmotility
- Neureally mediated hypotension, mitral valve prolapse
- Noncardiac chest pain, dyspnea due to respiratory mm. dysfunction
- Interstitial cystitis, female urethral syndrome, vulvar vestibulitis, vulvodynia

Restless legs syndrome
Fibromyagianess
Central Sensitization

- Fibromyalgia
- Chronic fatigue Syndrome
- Posttraumatic Stress Disorder
- Female Urethral Syndrome/Interstitial Cystitis
- Primary Dysmenorrhea
- Functional Gastrointestinal Disorders
- Tension Type Headaches
- Migraines
- Temporomandibular Disorders
- Multiple Chemical Sensivities
- Restless Legs Syndrome
- Myofascial Pain Syndromes
Abnormalities During Restorative (non-REM) Sleep in Fibromyalgia Patients

Psychological findings in Fibromyalgia

• Only 25% to 35% have a current psychiatric (DSM) diagnosis, most often major depression.
• As in most other chronic disorders, most patients report some mood disturbances such as feeling anxious or depressed.
The Clinical Challenge: Patient Cycling Contributes to Underdiagnosis

- ~5 million individuals with FM symptoms
- 94% present to HCP
  
  In a practice with 30 patients/day, 1-3 may have FM symptoms

- Dx with comorbid condition
- Cycling (average of 5 years)
- Tx but then re-present

- Overall diagnosis rate is low

- No definitive laboratory tests for diagnosis
- FM not suspected early in "cycling" process

- Multiple symptoms
- Confounding comorbidities
- Symptom descriptions do not always facilitate diagnosis

HCP=health care provider.
The Pain Matrix
Pathophysiology of FM

• Central amplification is a leading theory of FM pathophysiology
• fMRI data support FM as a disorder of central pain amplification
  – Areas activated by high-intensity stimuli in control patients were activated by low-intensity stimuli in patients with FM
• Elevated pain neurotransmitters (eg, substance P, glutamate) seen in patients with FM
• May contribute to pain amplification
FM: An Amplified Pain Response

Subjective Pain Intensity

Stimulus Intensity

Hyperalgesia
(eg, when a pinprick causes an intense stabbing sensation)

Allodynia
(eg, hugs that feel painful)

Pain in FM

Pain amplification response

Normal Pain Response

Central Amplification: Leading Theory for Abnormal Pain Processing in FM

Perceived pain

Ascending input

Descending modulation

Nociceptive afferent fiber

Pain stimuli

Normal Pain Processing

Perceived pain (hyperalgesia/allodynia)

Increased release of glutamate and substance P

Pain amplification

Decreased release of norepinephrine and serotonin

Induction of central amplification leading to abnormal pain processing

Minimal stimuli

Pain Processing in FM

fMRI Study Supports the Amplification of Normal Pain Response in Patients With FM

Patients with FM experienced high pain with low-grade stimuli

Overlapping regions of brain activation were seen in patients with FM after low pain stimuli and in normal subjects after high pain stimuli

- Red: Activated by low-intensity stimulus in FM patients
- Blue: Activated only by high-intensity stimulus in controls
- Yellow: Area of overlapping activation

fMRI=functional magnetic resonance imaging.

The brain as a network. (A) The brain can be represented as a network where the nodes correspond to constitutive elements, or nodes (voxels from MRI, neurons, etc.), and these nodes form links based on some type of interaction between nodes. Complex networks also have a tendency to exhibit a modular topology, where links are concentrated within modules. The presence of chronic pain may alter the properties of these modules through changes in inter- or intra-modular interactions, through peripheral and central mechanisms.

Pain Modulation: Serotonin and Norepinephrine

- Pain is associated with increased excitation and decreased inhibition of ascending pain pathways\(^1,2\)
- Descending pathways modulate ascending signals\(^1,2\)
- Norepinephrine (NE) and serotonin (5-HT) are key neurotransmitters in descending inhibitory pain pathways\(^1,2\)
- Increasing the availability of NE and 5-HT may promote pain inhibition centrally\(^1\)

**Parasympathetic**
- Constricts pupil
- Stimulates salivation
- Inhibits heart
- Constricts bronchi
- Stimulates digestive activity
- Stimulates gallbladder
- Contracts bladder
- Relaxes rectum

**Sympathetic**
- Dilates pupil
- Inhibits salivation
- Relaxes bronchi
- Accelerates heart
- Inhibits digestive activity
- Stimulates glucose release by liver
- Secretion of epinephrine and norepinephrine from kidney
- Relaxes bladder
- Contracts rectum

**Sympathetic ganglia**
- Cervical
- Thoracic
- Lumbar
Emerging Concept:
FM Has Complex Interactions with Endocrine and Immune Systems Regulating Emotion

Genes Involved in Pain Perception and Modulation.

## Table 1. Genes Involved in the Pain Genome

<table>
<thead>
<tr>
<th>Type</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cytokines</td>
<td><em>IL6, IL10, TNF</em></td>
</tr>
<tr>
<td>Enzymes</td>
<td><em>COMT, GCH1, CYP2D6</em></td>
</tr>
<tr>
<td>Ion channels</td>
<td><em>KCNS1, CACNG2, CACNA2D3</em></td>
</tr>
<tr>
<td>Receptors</td>
<td><em>OPRM1, ADRA2, DRD2</em></td>
</tr>
<tr>
<td>Transporters</td>
<td><em>DAT1, 5HTT, ABCB1</em></td>
</tr>
</tbody>
</table>

## Table 2. Genes That May Reduce or Protect From Pain

<table>
<thead>
<tr>
<th>Gene</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>COMT</em></td>
<td>encoding catechol-O-methyltransferase</td>
</tr>
<tr>
<td><em>GCH1</em></td>
<td>encoding guanosine triphosphate cyclohydrolase 1</td>
</tr>
<tr>
<td><em>MC1R</em></td>
<td>encoding melanocortin-1 receptor</td>
</tr>
<tr>
<td><em>OPRM1</em></td>
<td>encoding mu-opioid receptor</td>
</tr>
<tr>
<td><em>TRPV1</em></td>
<td>encoding transient receptor potential cation channel</td>
</tr>
</tbody>
</table>
Risk for chronic pain

Hardware at birth
- Gender, genotype and epigenetic profile

Environmental influences
- Acute injury or disease at critical developmental periods
- Stressful life events

Gene × environment interactions
- Personality and psychology (for example, pessimism, neuroticism, anxiety, catastrophizing, reward bias)

Innate mechanisms
Acquired mechanisms
Brain vulnerable networks
The Patient Is at the Center of the Fibromyalgia Treatment Program

- primary care physician/rheumatologist

PATIENT

- Physical medicine and rehabilitation
- Support system
  - family
  - mental health professionals

Source: Panel Consensus.
Core Treatment of FM

1. Confirm diagnosis
   - Identify important system domains, their severity, and level of patient function
   - Evaluate for comorbid medical and psychiatric disorders
   - Assess psychosocial stressors, level of fitness, and barriers to treatment
   - May require referral to a specialist for full evaluation
   - Provide education about FM
   - Review treatment options; initiate monotherapy on the basis of the patient’s presentation and evidence-based guidelines

Nonpharmacologic Strategies: Evidence of Efficacy

Strong Evidence
- Exercise
  - Physical and psychological benefits
  - May increase aerobic performance and tender-point pain pressure threshold, and improve pain
  - Efficacy not maintained if exercise stops
- Cognitive-behavioral therapy
  - Improvements in pain, fatigue, mood, and physical function
  - Improvement often sustained for months
- Patient education/self-management
  - Improves pain, sleep, fatigue, and quality of life
- Combination (multidisciplinary therapy)

Modest Evidence
- Strength training
- Acupuncture
- Hypnotherapy
- EMG biofeedback
- Balneotherapy (medicinal bathing)
- Transcranial electrical stimulation

Weak Evidence
- Chiropractic
- Manual and massage therapy
- Ultrasound

No Evidence
- Tender-point injections
- Flexibility exercise

Exercise Therapies for Fibromyalgia

• Aerobic exercise - cardiovascular fitness
  - Water exercise
  - Walking
  - Cycling
  - Low impact aerobics

• Regular stretching, calisthenics
Change in Function Scores for Exercise and Control Patients With FM

Possible neural pathways of cognitive pain modulation. Cognitive modulations of pain are related to activation of prefrontal brain areas such as the dorsolateral prefrontal cortex (DLPFC), ventrolateral prefrontal cortex (VLPFC), and to the anterior cingulate cortex (ACC); shown in orange. These regions may modulate activation in afferent pain regions in the cortex (ACC, primary- and secondary somatosensory cortex, insula and thalamus), as well as the periaqueductal gray (PAG) and dorsal horns of the spinal cord.
Efficacy of Multicomponent Treatment in FM: A Meta-analysis of RCT

Strong evidence that Rx reduced pain, fatigue, mood, and HRQOL in short but not long term.

FM: New Treatment Approaches

- Patient subsets are treated differently
- Combinations of current agents
- Combine nonmedicinal with drug therapies
- Newer analgesics
- Targeted drug treatments
- Multidisciplinary programs
Placebo and nocebo modulation of pain. Whereas placebo suggestions activate mu-opioid neurotransmission which inhibits pain, nocebo suggestions induce anxiety which activates CCK-A and/or CCK-B receptors that, in turn, enhance pain.

When words are painful: Unraveling the mechanisms of the nocebo effect

Neuroscience, 147, 2007, 260 - 271
## FM Pharmacologic Therapies

<table>
<thead>
<tr>
<th>Evidence Level</th>
<th>Therapeutic Agents</th>
</tr>
</thead>
</table>
| **Strong Evidence** | Antidepressants  
- Tricyclic compounds (amitriptyline, cyclobenzaprine)  
- Norepinephrine reuptake and serotonin-norepinephrine reuptake inhibitors (milnacipran, duloxetine)  
- Pregabalin |
| **Modest Evidence** | Tramadol  
- Gabapentin  
- Selective serotonin reuptake inhibitors  
- Gamma-hydroxybutyric acid  
- Dopamine agonists (pramipexole) |
| **Weak Evidence** | Growth hormone, 5-hydroxytryptamine, S-adenosyl-L-methionine (SAMe) |
| **No Evidence** | Opioids, corticosteroids, nonsteroidal anti-inflammatory drugs, benzodiazepine and nonbenzodiazepine hypnotics, guaifenesin, thyroid hormone |

FM: Newer Pharmacologic Therapies

- Alpha-2-delta ($\alpha_2\delta$) ligands
  - Pregabalin (approved by the US Food and Drug Administration [FDA] in 2007 for the management of FM)
  - Gabapentin *

- Serotonin-norepinephrine reuptake inhibitors
  - Duloxetine (approved by the FDA in 2008 for the management of FM)
  - Milnacipran (approved by the FDA in 2009 for the management of FM)

*This information concerns a use that has not been approved by the FDA.
Pregabalin and Gabapentin

- Bind to $\alpha_2\delta$ subunit of voltage-gated calcium channels of neurons
- Reduce calcium influx at nerve terminals and therefore inhibit release of neurotransmitters, such as glutamate & substance P

Pregabalin 14-Week Fixed-Dose FM Trial

Mean change from baseline

Week

Placebo (n=184)
Pregabalin 300 mg (n=183)
Pregabalin 450 mg (n=190)
Pregabalin 600 mg (n=188)

P<.05 all 3 doses vs placebo except 300 mg/day at Week 11

Baseline mean = 6.7 (moderate-to-severe pain)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose, mg/d</th>
<th>No. of Individual RCTs</th>
<th>No. of Participants</th>
<th>Patients Achieving Outcome, No./Total (%)</th>
<th>Risk Ratio (95% CI)</th>
<th>NNT (95% CI)&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Painful diabetic neuropathy</strong></td>
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<tr>
<td>Gabapentin&lt;sup&gt;b&lt;/sup&gt;</td>
<td>600-3600</td>
<td>4</td>
<td>829</td>
<td>188/467 (40) 83/362 (23)</td>
<td>1.8 (1.4-2.2)</td>
<td>5.8 (4.3-9.0)</td>
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<td></td>
<td>400</td>
<td>2</td>
<td>412</td>
<td>97/274 (35) 35/138 (25)</td>
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<td>10 (5.2-120)</td>
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<td>Lamotrigine&lt;sup&gt;b&lt;/sup&gt;</td>
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<td>3</td>
<td>773</td>
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<td>645</td>
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<td>11 (6.1-54)</td>
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<td></td>
<td>600</td>
<td>4</td>
<td>1005</td>
<td>278/608 (46) 118/397 (30)</td>
<td>1.5 (1.3-1.8)</td>
<td>6.3 (4.6-10)</td>
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<td><strong>Postherpetic neuralgia</strong></td>
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<tr>
<td>Gabapentin&lt;sup&gt;b&lt;/sup&gt;</td>
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<td>3</td>
<td>892</td>
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<td>7.5 (5.2-14)</td>
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<td></td>
<td>300</td>
<td>3</td>
<td>535</td>
<td>79/263 (30) 30/272 (11)</td>
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<td>5.3 (3.9-8.1)</td>
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<td></td>
<td>600</td>
<td>3</td>
<td>551</td>
<td>109/276 (39) 39/275 (14)</td>
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<td>4.0 (3.1-5.5)</td>
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<td><strong>Central neuropathic pain&lt;sup&gt;c&lt;/sup&gt;</strong></td>
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<td></td>
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<tr>
<td>Pregabalin</td>
<td>600</td>
<td>2</td>
<td>176</td>
<td>22/89 (25) 6/87 (7)</td>
<td>3.6 (1.5-8.4)</td>
<td>5.6 (3.5-14)</td>
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<tr>
<td><strong>Fibromyalgia</strong></td>
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<tr>
<td>Pregabalin</td>
<td>300</td>
<td>4</td>
<td>1374</td>
<td>147/685 (21) 99/689 (14)</td>
<td>1.5 (1.2-2.9)</td>
<td>14 (9.0-33)</td>
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<td>450</td>
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<td>1376</td>
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<td>1.6 (1.3-2.1)</td>
<td>11 (7.1-21)</td>
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</tbody>
</table>

Abbreviations: NNT, number needed to treat; RCT, randomized clinical trial.

<sup>a</sup> NNT was not calculated when risk ratio included 1, showing no significant benefit over placebo.

<sup>b</sup> Analyses were performed using a particular maximum daily dose except for lamotrigine, where doses between 200 mg and 400 mg were allowed, and for gabapentin, where a range of daily doses were used.

<sup>c</sup> Central neuropathic pain is neuropathic pain following spinal cord injury or stroke. For inclusion, results were required from 2 studies with at least 200 participants in total, but this was waived for central neuropathic pain because of the importance and difficulty of performing studies on this condition.
Serotonin-Norepinephrine Reuptake Inhibitors

- Efficacy of serotonin-norepinephrine reuptake inhibitors in chronic pain conditions, independent of their effects on mood
  - Thought to be due to effects on serotonin- and norepinephrine-mediated descending pain-inhibitory pathways in the brain and spinal cord
- Agents with dual serotonin and norepinephrine activity may have more consistent benefits in relief of persistent pain
Milnacipran: Improvements in the PED Morning-Recall Pain Score Over Time

PED = patient experience diary
*P < .01; †P < .001

Analgesics in FM

- Tramadol (Ultram®, Ultracet®)
  - Opioid receptor and serotonin receptor agonist with monoamine reuptake inhibition
  - Effective in 3 randomized controlled trials (RCTs)
- N-methyl-D-aspartate (NMDA) receptor antagonists
  - Ketamine: no controlled studies
  - Dextromethorphan: 1 study, minimal efficacy
- Opioids: no controlled studies
- NSAIDs, COX-2: used as adjuncts with little evidence
A Prospective, Long-term FM Follow-up Study

- 65% believed that FM was better than when first diagnosed
- 55% felt well or very well
- No patients developed a connective tissue disease or another diagnosis
- Only 9% had to leave a job because of FM
- 71% believed that FM interfered little if at all with work

Heterogeneity Within the Fibromyalgia Population: Theoretical Implications of Variable Tender Point Severity Ratings

HILARY D. WILSON, TERENCE W. STARZ, JAMES P. ROBINSON and DENNIS C. TURK

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Abstract

Objective. The American College of Rheumatology (ACR) tender point (TP) criterion is used in diagnosing fibromyalgia syndrome (FM). There has been little research investigating patterns of positive TP. We investigated response patterns of TP in a sample of patients with FM.
What Are the Typical Outcomes in FM?

- Most patients have chronic, persistent symptoms; complete remission not rare
- Most patients continue to work, but 10%-15% are work disabled
- There is often an adverse impact on work and leisure activities
- Duration of time without a diagnosis adversely affects outcome
- Outcome much better in community vs tertiary care
Evolution of Thinking Regarding Fibromyalgia

American College of Rheumatology (ACR) Criteria

- Discrete illness
- Focal areas of tenderness
- Psychological and behavioral factors nearly always present and negative

- Chronic widespread pain
- Tenderness in ≥ 11 of 18 tender points

- Final common pathway
- Part of a larger continuum
- Many somatic symptoms, diffuse tenderness
- Psychological and behavioral factors play roles in some individuals

Anterior  Posterior

Clauw
### Perception versus Reality in Fibromyalgia Diagnosis

<table>
<thead>
<tr>
<th>Perception</th>
<th>Reality</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;It's not a real condition, just a wastebasket diagnosis&quot;</td>
<td>It was classified in 1990 by the ACR as a discrete disorder</td>
</tr>
<tr>
<td>&quot;It's just the new trend&quot;</td>
<td>It has a 200-year medical history</td>
</tr>
<tr>
<td>&quot;It's all in the patient's head&quot;</td>
<td>Studies show no psychiatric Dx in most patients</td>
</tr>
<tr>
<td>&quot;Patients with fibromyalgia have all the same personality traits&quot;</td>
<td>Patients vary; similarities related to the impact of chronic symptoms on personality</td>
</tr>
<tr>
<td>&quot;Patients just have to learn to live with it&quot;</td>
<td>Providing patients with diagnosis and treatment helps them cope with symptoms</td>
</tr>
</tbody>
</table>

FIBROMYALGIA

- Cognitive Impairment 'Fibro Fog'
- Muscular Pain
- Morning Stiffness
- Chronic Headaches
- Dizziness
- Irritable Bowel Syndrome
- Insomnia
- Chest Pain
- 18 Tender body Points
- Blurry Vision
- Sensitivity To Light / Sound
- Fatigue
- Body Aches
- Feeling Cold
- Dry Eyes
- Weight Gain
- Nausea
- Anxiety & Depression