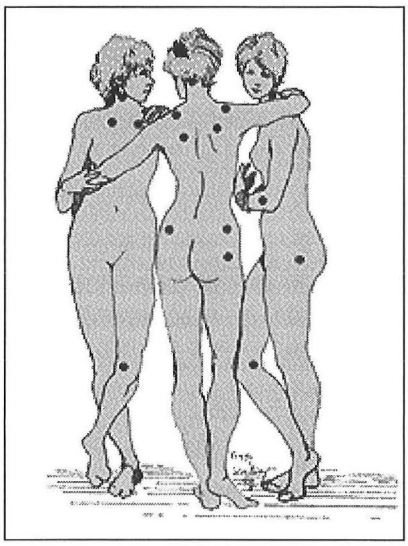
Fibromyalgia and the Social Construction of Disease



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American College of Rheumatology

1990 criteria for the classification of Fibromyalgia

1. History of widespread pain.

Definition. Pain is considered widespread when all of the following are present: pain in the left side of the body, pain in the right side of the body, pain above the waist, and pain below the waist. In addition, axial skeletal pain (cervical spine or anterior chest or thoracic spine or low back) must be present. In this definition, shoulder and buttock pain is considered as pain for each involved side. "Low back" pain is considered lower segment pain.

2. Pain in 11 of 18 tender point sites on digital palpation.

Definition. Pain, on digital palpation, must be present in at least 11 of the following 18 sites:
Occiput: Bilateral, at the suboccipital muscle insertions.
Low cervical: bilateral, at the anterior aspects of the intertransverse spaces at C5-C7.
Trapezius: bilateral, at the midpoint of the upper border.
Supraspinatus: bilateral, at origins, above the scapula spine near the medial border.
Second rib: bilateral, at he second costochondral junctions, just lateral to the junctions on upper surfaces.
Lateral epicondyle: bilateral, 2 cm distal to the epicondyles.
Gluteal: bilateral, in upper outer quadrants of buttocks in anterior fold of muscle.
Greater trochanter: bilateral, posterior to the trochanteric prominence.
Knee: bilateral, at the medial fat pad proximal to the joint line.

Digital palpation should be performed with an approximate force of 4 kg.
For a tender point to be considered "positive" the subject must state that the palpation was painful. "Tender" is not to be considered "painful."

* For classification purposes, patients will be said to have fibromyalgia if both criteria are satisfied. Widespread pain must have been present for at least 3 months. The presence of a second clinical disorder does not exclude the diagnosis of fibromyalgia.

Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: report of the multicenter criteria committee. Arthritis Rheum 1990;33:160-72.

In 1990 the American College of Rheumatology published criteria for the classification of fibromyalgia [1]. The naming of a disorder and its endorsement by a medical organization created the necessary authenticity required for patients and physicians to make this enigmatic syndrome a tangible entity. Fibromyalgia was defined as a syndrome of widespread pain associated with characteristic tender points. Though not part of the formal criteria, patients were noted to experience fatigue and sleep disturbances. There is also considerable overlap with disorders such as irritable bowel syndrome, interstitial cystitis, migraine headaches and chronic fatigue syndrome.

Fibromyalgia is a controversial disorder

Despite entering the medical lexicon, fibromyalgia remains a controversial disorder. Its very name, suggesting a pathophysiologic insight (which is considered unsubstantiated), has been widely criticized. Physicians are polarized into those that accept fibromyalgia as a bona fide disease and those that regard it as the by-product of physician and societal sanctioning of universal psychosocial stress [2;3]. Physicians are clearly ambivalent in their acceptance of a disease that is largely diagnosed by self-report, for which there is no reliable laboratory test or imaging procedure and for which specific treatment does not exist. Others are distressed by the burgeoning development of a parallel informal health-care system that excludes the traditional model of health care delivery. Patients are seen as caught in a self-perpetuating loop of unscientific information on the Internet, consuming unproven nutriceutical remedies and turning to litigation to validate their disease.

It is the purpose of this presentation to discuss the basis of this contention and the broad insight fibromyalgia provides into the nature of the physician-patient-societal interaction. It is the assertion of the author that such interaction is much more pervasive than physicians are willing to acknowledge.

Case presentation

Loren Mills is a 53-year-old Caucasian woman who is referred by her gastroenterologist for suspected fibromyalgia. She complains of the insidious onset of low back pain several years ago, that over the last two years, has spread to her neck and shoulders. She now hurts in all her muscles and joints. She has undergone three back operations without improvement. Additionally, she suffers from hyperlipidemia, hypertension, migraine headaches and depression. She admits to insomnia and has non-restorative sleep. She experiences several hours of morning stiffness in her joints and has subjective intermittent swelling of the hands and wrists. Ms. Mills has had several driving accidents secondary to panic attacks. She last worked as a bartender but now receives social security disability payments. She is divorced but living with her ex-husband. Both her children also suffer from migraine headaches and back problems. Additionally the daughter has depression and has been diagnosed with fibromyalgia. Recent stressors include the death of two of her dogs attributed to poisoning and parvovirus infection in their five puppies.

Review of systems is notable for weight gain, fatigue, weakness, memory loss, burred vision, frequent sore throats and dysphagia. She has noted swollen and tender lymph nodes in her neck. Ms. Mills underwent an extensive, unrevealing cardiac evaluation for chest pain. Additional complaints include persistent diarrhea, heartburn and chronic nausea. She also

admits to urinary frequency, vaginal dryness and dyspareunia.

She is allergic to penicillin, Imitrex, Biaxin, Cipro, DHE 45, aspirin, Motrin and Toradol. Current medications include Prozac, Fioricet, Ambien, Valium, Plavix, verapamil, Midrin, Cytotec, Prilosec, Lipitor, colesevelam and Premarin cream.

Upon physical examination, she appeared anxious. She had normal muscle strength, and normal range of motion of the joints. There was no synovial thickening, but she had diffuse tenderness of the joints. All 18 of the designated fibromyalgia points were tender.

This patient meets criteria for fibromyalgia [1] as well as for chronic fatigue syndrome [4].

Clinical features of fibromyalgia and chronic fatigue syndrome

Fibromyalgia is a clinical syndrome characterized by widespread chronic muscle pain most prominent in the axial region but also involving the anterior chest wall and limbs. The pain is variable in intensity and worsens with activity. Patients cannot distinguish well between myalgia and arthralgia and often describe their joints as being swollen. Concurrently reported uncomfortable sensations are described as burning, numbness, tingling and a sense of heaviness of the limbs.

Fatigue is very frequent (90%) and may be the presenting complaint. Patients report difficulty falling off to sleep, frequent awakenings and feeling unrefreshed the next morning. Mood disturbance, cognitive impairment, headache, Raynaud's phenomenon and pre-syncope are also very prevalent. Some patients meet formal criteria for depression.

Many individuals have concurrent irritable bowel syndrome, chronic fatigue syndrome, migraine headaches and interstitial cystitis.

Physical examination is largely normal except for the finding of characteristic tenderness at defined trigger points. Laboratory studies, likewise, are within normal limits.

Fibromyalgia is strikingly more common in women and appears to linearly increase with age with a prevalence of 2% at age 20 and 8% at age 70. Peak presentation is in the fourth and sixth decades of life. In approximately half the cases, onset is attributed to trauma or a flu-like illness.

In chronic fatigue syndrome, disabling persistent fatigue is the primary complaint. It is unrelieved by rest. Concurrent symptoms (by definition) must additionally include at least four of the following symptoms: sore throat, tender lymph nodes, muscle pain, multi-joint pain, headaches, unrefreshing sleep and post-exertional malaise [4].

There is substantial overlap between these two disorders suggesting that both disorders may represent the same condition [5;6]. Aaron and Buchwald reviewed selected studies that compared physiologic markers in at least two of the following 12 "unexplained clinical conditions": fibromyalgia, chronic fatigue syndrome, irritable bowel syndrome, temporomandibular disorder, multiple chemical sensitivity, tension and migraine headaches, interstitial cystitis, chronic nonbacterial prostatitis, chronic pelvic pain, chronic low back pain and post-concussion syndrome. In studies that evaluated physical findings, tender points were the most

common shared clinical feature. If studies evaluated pain and sleep abnormalities, patients did not differ greatly from each other in pain tolerance and reductions in pain thresholds [5].

Is fibromyalgia a new disorder or is it a new name for an old disorder?

In 1869 George Beard, described neurasthenia, or nervous exhaustion [7]. Patients reported "heaviness and vague aching of the loins and sometimes of the whole body". Beard attributed these symptoms to the stresses of modern life. Similar symptoms and descriptions of overwhelming fatigue are almost universally reported by patients with fibromyalgia and chronic fatigue syndrome.

Reports of neurasthenia began to diminish by the early 1900s and a new fatigue syndrome labeled epidemic neuromyasthenia or benign myalgic encephalomyelitis appeared in the literature in the mid 1930s. The epidemic nature of this syndrome suggested either an infectious agent or mass hysteria. The adoption of the infection hypothesis resulted in the label of postviral syndrome [8].

In the 1980s there was a resurgence of interest in the post-infectious hypothesis with chronic Ebstein-Barr virus infection implicated in the pathogenesis of what was soon termed the chronic fatigue syndrome [9].

The term fibromyalgia was preceded by the concept of fibrositis first used by Gowers in 1904 [10] to describe non-mechanical back pain. In 1938, Kellgren described the phenomenon of pain referred from muscle [11]. Prior to 1980 fibrositis referred to localized disorders (now replaced by the term "myofascial pain syndrome") and was only recently used to indicate the widespread pain disorder for which the name fibromyalgia was suggested by Hench in 1976 [12] and subsequently adopted by the American College of Rheumatology in 1990 [1].

Fibromyalgia is clearly not a new disease. It is simply a new term for an old condition that has been assigned many different names. Its new designation is defended by its creators as a "clinical construct that allows physicians and others to describe and communicate to themselves a definition of one kind of pain syndrome" [13].

Pathophysiology of fibromyalgia: evolving views

Since muscle pain is a central feature of fibromyalgia, the earliest studies of this disorder focused on attempts to demonstrate pathologic changes in muscle. This endeavor has been comprehensively reviewed by Simms [14]. Following the descriptions of fibrositis by Gowers in 1904 [10], Stockman performed biopsy studies of palpable tender nodules in patients with fibrositis and described "inflammatory hyperplasia" in excisional biopsy specimens. Two subsequent studies could not confirm these findings and a review of Stockman's original specimens by Collins in 1940 found no evidence of inflammation. Similarly, contemporary muscle biopsy studies have revealed either normal findings or nonspecific ultrastructural changes. Studies of muscle metabolism and MR spectroscopy have also failed to demonstrate abnormalities in muscle metabolism [14].

Is fibromyalgia a psychiatric disorder?

With the failure to find local muscle pathology to explain the widespread muscle pain and given the high frequency of either a history of, or concurrent depression, it is reasonable to examine the premise that fibromyalgia is a form of either overt or

subsyndromal depression. Additionally, symptoms of major depression like sleep disorder, depressed mood, poor concentration and fatigue are very prevalent in fibromyalgia, suggesting that these two diseases are closely related.

It is not uncommon to find articles and textbook chapters published in the recent past [15] devoted to psychogenic rheumatism and to find, in the contents, descriptions that seem to match those of modern day fibromyalgia patients: a dramatic urgency to be seen by the doctor; a written list of complaints; a large volume of previous investigations brought to the first clinic visit. This prevailing view of fibromyalgia as a psychogenic or hysterical disorder was widely held until the early 1980's when more formal studies of the relationship between fibromyalgia and psychiatric disorders were conducted [16].

In 1982 Payne administered the Minnesota Multiphasic Personality Inventory (MMPI) to 30 hospitalized patients with fibromyalgia and found higher MMPI scores in fibromyalgia patients when compared with patients with arthritis [17]. Subsequent studies [18;19] found similar findings, suggesting that patients with fibromyalgia have psychiatric disturbance. A closer analysis of these studies indicates that fibromyalgia patients scored higher on the hypochondriacal and hysteria scales but not on the depression scale [16]. Smythe suggested that the MMPI will rate any patient with chronic pain high on the hysteria and hypochondriasis scales [20]. Studying patients with rheumatoid arthritis, Pincus found that elevated MMPI scores for hypochondriasis, depression and hysteria correlated with disease activity [21]. Interestingly, testing fibromyalgia patients from general practice, Clarke did not find higher rates of psychologic disturbance [22], suggesting that psychologic disturbance in patients with fibromyalgia is more common in referral-based practices when compared with patients in the community.

Patients with fibromyalgia are more likely to report a personal history of depression (50%-70%) yet current major depression is found in not more than 36% of patients [23]. A prospective study of 175 women with self-reported pain, designed to examine etiologic factors in the onset of fibromyalgia (which developed in 25% of this cohort after a period of 5.5 years), revealed that self-reported depression at baseline was the single strongest predictor (six-fold) of new-onset fibromyalgia [24]. Taken together, these data suggest that depression and fibromyalgia are associated, but the nature of the association and the temporal relationship are unclear. The prevailing view is that fibromyalgia and major depression share a common etiologic abnormality [25]. It is also increasingly believed that the greater frequency of depression in fibromyalgia patients in referral-based practices versus fibromyalgia patients in the community reflect differences in health-seeking behaviors [26-28].

Attention has also turned to fibromyalgia as a variant of somatization disorder since patients with this disorder have multiple somatic complaints that suggest organic disease, but none is found. These include severe fatigue, abdominal pain, diarrhea, headaches, jaw pain, paraesthesia, Raynaud's phenomenon, dysuria and dizziness, amongst others. Some of these symptoms translate into definitions of related syndromes like irritable bowel syndrome.

Most patients with fibromyalgia would not satisfy diagnostic criteria for somatization

disorder [29], leading to the notion that these patients may have a subsyndromal somatization state [30]. Critics of the somatization disorder viewpoint draw attention to the circular nature of the definition of somatization disorder: "a psychiatric diagnosis, that depends on the presence of physical symptoms that suggest organic disease and are not explained by a general medical condition, would become a *non*psychiatric diagnosis once the general medical condition adequately explains the symptoms" [31].

Fibromyalgia as a chronic pain disorder

One of the prevailing views of the pathophysiology of fibromyalgia maintains that it is a disorder of abnormal central sensory processing and points to an accumulating body of evidence suggesting that this is the fundamental phenomenon in this disease [31-35].

With the failure to find muscle pathology in fibromyalgia and the realization that most patients with fibromyalgia do not have a formal psychiatric diagnosis, attention has been directed to the neurobiology of chronic pain as the key to understanding the cause of fibromyalgia. This shift in focus was facilitated by a better understanding of the pathophysiology of chronic pain. Additionally, studies of the epidemiology of chronic pain have correlated well with the epidemiology of fibromyalgia. A survey of over 2000 adults in England revealed the surprising finding that 11.2% of these "non-patients" reported chronic widespread pain [36]. When this subgroup was examined, 21.5% had 11 or more tender points (criteria-positive fibromyalgia), while 63.8% had between 1 and 10 tender points. Interestingly, the number of tender points correlated best with depression, fatigue and poor sleep. In a similar population-based study in Kansas, Wolfe found that widespread pain was more common in women and appeared to increase in prevalence with age, reaching 23% by the seventh decade [37].

Both the higher prevalence of widespread pain in the population when compared with the prevalence of fibromyalgia, and the quantitative difference in tender points, suggests that fibromyalgia lies at one end of a continuous spectrum of chronic pain. Recalling that the presence of multiple tender points correlates better with symptoms of depression, fatigue and sleep disorder, a picture emerges that suggests that subsyndromal fibromyalgia achieves syndromic expression (hence, defined recognition) when fatigue, sleep disorder and depression are added to a background of widespread chronic pain.

The evolving concept of chronic pain as resulting from abnormal centrally-mediated sensory processing affected by a complex integration of noxious stimuli, affective traits and cognitive factors has allowed the emergence of a model that provides avenues for integration of neuroendocrine, psychosocial and genetic theories of fibromyalgia.

Studies of juvenile fibromyalgia have reported familial aggregation of fibromyalgia and related chronic pain syndrome, suggesting a biologic basis, but investigators have been unable to exclude shared environmental variables [38].

The concept of non-nociceptive pain (NNP) has emerged from observations that persistent stimulation of peripheral nerves can lead to a disproportionate augmentation of central sensory processing. NNP is pain elicited by stimulation of fibers that usually relay non-painful signals to the spinal cord. These normally non-noxious

stimuli are subverted by abnormal central processing resulting in the experience of pain.

Several studies of chronic pain in fibromyalgia were recently reviewed by Bennett [31] and have established the following findings in fibromyalgia patients when they are compared with controls: 1) dolorimetry studies in fibromyalgia show a lower pain threshold; 2) with isometric muscle contraction, the pain threshold in fibromyalgia increases rather than showing the expected decrease seen in controls; 3) somatosensory-induced potentials recorded using skull electrodes demonstrate increased amplitude following laser stimulation of skin. Additional studies using neuroimaging techniques, reviewed by Bradley [39], also provide indirect evidence of abnormal central sensory pain processing by demonstrating abnormalities in cerebral blood flow in the thalamus and caudate nuclei in patients with fibromyalgia.

Fibromyalgia and the neuroendocrine axis

The hypothalamic-pituitary-adrenal axis (HPA) is the primary endocrine stress axis in man. It has evolved as an adaptational response to metabolic, infectious, inflammatory and emotional stressors responding to both endogenous and exogenous stimuli. An emerging concept is that both fibromyalgia and chronic fatigue syndrome can be viewed as consequences of subversion of this primitive adaptational response by abnormal stress activation in vulnerable individuals.

Stress-mediated activation of the HPA axis and sympathetic nervous system functions to ready the organism for danger (*flight or fight*). Activation leads to a state of alertness, focused attention, vigilance and aggression. There is simultaneous inhibition of vegetative functions: growth, reproduction and restraint of the immune response. Energy is diverted away from these functions to the CNS and critical musculature. Corticotropin-releasing hormone (CRH), a peptide hormone produced in the hypothalamus, is the leading mediator of HPA axis activation. As implied by its name, CRH by way of adrenocorticotrophic hormone (ACTH) stimulation, increases adrenal cortisol production. Additionally CRH also appears to increase somatostatin levels which in turn inhibit the secretion of growth hormone and thyroid stimulating hormone. CRH will also stimulate the locus ceruleus neurons, causing release of norepinephrine in the brain. CRH secretion may be stimulated by pain, emotional stress, serotonin and its precursors like tryptophan.

Neuroendocrine studies of the HPA axis [32;40;41] in patients with fibromyalgia have shown the following abnormalities: 1) elevated cortisol levels with a flattened diurnal pattern that are not suppressed by dexamethasone administration; 2) low 24-hour urinary free cortisol suggesting elevated cortisol secretion during the day and suppressed secretion at night; 3) markedly enhanced ACTH release when CRH is injected, but with no increase in cortisol levels, suggesting adrenal hyporesponsiveness.

These hormonal perturbations have been interpreted to represent chronic CRH hyperactivity driven by stress and pain. Secondary effects include changes in the set points of other hormonal axes like growth hormone, gonadotropin and thyroid stimulating hormone. Interestingly, neuroendocrine findings in the chronic fatigue syndrome suggest hyposecretion of CRH.

While the significance and cause-effect relationship of these neuroendocrine abnormalities is still a work in progress, it serves as an attractive model to explain the confluence of pain and stress in the pathogenesis of fibromyalgia. Whether somatic symptoms like post-exertional fatigue reported in fibromyalgia are caused by these abnormalities remains unclear although observations, such as of increased gut smooth muscle tone secondary to stress, suggest that the link between somatic complaints and central nervous system changes is bidirectional [42].

The prevailing view of the pathogenesis of fibromyalgia

Any unified hypothesis of the cause of fibromyalgia would begin with the disclaimer that no single pathologic abnormality can explain the epidemiology and clinical features of this disorder. What is increasingly agreed upon is that the disorder often begins as a localized pain syndrome, often in the axial skeleton. If a background of abnormal central sensory perception exists, a generalized pain syndrome may ensue. Concurrent HPA axis stimulation may serve as a modulator of ongoing central pain sensitization and serves as a link to psychosocial stressors that may create a complex, interactive, self-amplifying loop of worsening symptoms and increasing stress.

Summary of the current concept of fibromyalgia

Fibromyalgia is a complex syndrome characterized by widespread pain, fatigue, sleep disturbances and multiple somatic complaints. It was originally described as fibrositis but was given the designation of fibromyalgia in 1990 by the American College of Rheumatology. It has extensive overlap with chronic fatigue syndrome, itself a disorder that was originally called neurasthenia in the 19th century. Theories of the pathophysiology of fibromyalgia have moved from a disorder of soft tissues to one of chronic pain. Diagnosis is largely based on patient self-report but an increasing body of literature points to measurable phenomena in the central nervous system and HPA axis. Psychologic disturbances are common in patients with fibromyalgia but the temporal association with disease onset is still unclear. The search for a single pathophysiologic lesion appears fruitless since it is likely a multifactorial disorder.

The social construction of fibromyalgia: The birth of biomedicine

At the turn of the 18th century, medical thought underwent a radical change. Prior to this change, doctors were few in number and their practice was restricted to the care of the higher socioeconomic classes. The doctor-patient relationship was dominated by patients and their individual demands (symptoms). Attributed to the French Revolution and other contemporaneous political events, the practice of medicine shifted to the hospital. The study of pathologic anatomy overcame sociopolitical resistance and medicine changed from a study of symptoms to the precise mapping of signs and symptoms to observable pathologic lesions. Doctors found themselves caring for indigent, socially inferior (and less demanding/more passive) patients in a hospital setting. Simultaneously, the teaching of medicine became institutionalized. The doctor was now dominant in the doctor-patient relationship. Medicine acquired a precision that hitherto had only been the province of mathematics.

Michel Foucault, a French sociologist, has discussed this shift in the perception of scientific knowledge over time, in his

book, *The Birth of the Clinic: The Archeology of Medical Perception* [43]. Foucault suggests that diseases occupy a series of "spaces". Primary spatialization illustrates the means by which the disease is described and ordered as a concept. Secondary spatialization refers to the process by which the disease is given a place within the body. Tertiary spatialization explains the method by which the disease and the diseased individual are located within the societal body.

The concept of secondary spatialization, which explained disease as a definable lesion within the human body, gave genesis to biomedicine which remains the current basis of modern medical science. Disease after disease has been elegantly deciphered. Epidemiologic puzzles and seemingly disparate signs and symptoms have all made sense once the "lesion" was discovered.

However, not all diseases have yielded to this pathologic gaze. This is especially true of psychiatric diseases, where scrutiny of brain tissue has not revealed obvious pathology. Several psychiatric diseases have distinct neurotransmitter abnormalities which exhorts biomedicine to hold out the hope that lesions will be eventually discovered for all psychiatric disorders. Others argue that this hope is futile since the boundary of normal and abnormal behavior is often determined by prevailing social norms [44].

Symptoms, signs and illness behavior

The biomedical model of disease assumes that the presence of a pathologic lesion is revealed in two ways: *symptoms*, the patient's perception that body function is not normal, and *signs*, the physician's observation that signifies that an underlying lesion exists.

This model presumes that patients will behave according to the following expectation: the patient will cooperate with the biomedical model by not presenting with trivial symptoms, while not trivializing serious symptoms that require medical attention.

Community surveys suggest that the ratio of symptom episodes to consultation is much higher than one would expect. Banks studied a random sample of female patients aged between 20 and 44 years, in the United Kingdom. Women selected for the study completed a health diary for four weeks, and over 12 months their demand for general practitioner care was recorded [45]. The ratio of symptom episodes to consultations is shown below:

Headache	184:1
Backache	52:1
Emotional problem	46:1
Abdominal pain	28:1
Sore throat	18:1
Pain in chest	14:1

These data suggest that patients subject their symptoms to some form of evaluation prior to seeking medical advice. This evaluative process and the decision to seek medical help is encompassed by the term *illness behavior* which was first suggested by Mechanic and Volkart in 1960 [46].

Patients have to decide whether their symptoms are normal or abnormal. Widely prevalent symptoms like headache may often be viewed as normal. Chronic symptoms such as cough in smokers may be deemed normal. The actual doctor visit may be precipitated by one or more five social triggers as described by Zola [47]: 1) perceived interference with vocational or social activity; 2) perceived interference

with social or personal relations; 3) the occurrence of an interpersonal crisis; 4) failure of symptoms to resolve by the end of a self-determined arbitrary period; 5) coercion from friends or relatives (sanctioning).

In fibromyalgia, community surveys confirm that many more fibromyalgia *non-patients* than fibromyalgia *patients* exist, suggesting that many do not view their symptoms worthy of consultation [36;37]. A history of depression, rather than current depression, is common in fibromyalgia patients, suggesting that the history of depression is a marker for persons more likely to seek health care [26]. The "onset" of fibromyalgia following an interpersonal crisis has been well described in the literature signifying that a stressful life event, and not the disease itself, precipitates a consultation [27].

The costs and benefits of seeking health care

Ostensibly, patients seek health care for therapeutic benefits. Additionally, Parsons [48] has suggested that because of the strong position of social authority of the doctor, he or she is able to legitimize the illness. This allows the patient to ease from being a well person to being a patient. From this newly conferred "sick role", the patient can expect to gain two benefits while incurring two obligations (costs). The patient is temporarily excused from performing his or her normal social role and is not held responsible for his or her illness. The two obligations are: the patient must want to get well (the sick role is of temporary status) and the patient must cooperate with the treatment plan.

In this model, the doctor dominates and the patient is relatively passive and obedient. The doctor is acting in the patient's best interest and the patient cooperates in the context of a shared agenda in which the biomedical view of illness predominates.

While this model works well for most subacute illnesses, its utility in chronic illnesses is constrained by the inability of the patient to fulfill these two obligations: the sick role is not temporary and the lack of cure makes cooperating with treatment difficult to sustain. Similarly the model also fails when patients have multiple complaints without a readily identifiable lesion. Furthermore, the model fails when the patient does not accept a shared belief in this biomedical model of disease.

Good patients, great patients and bad patients

It is not only patients who have difficulty with such situations. Doctors are so heavily invested in this model that they view patients with chronic diseases or multiple complaints without readily identifiable lesions as "difficult patients". There is extensive countertransference as the responsibility of the disease is transferred back to the patient and the patient is increasingly held responsible for their symptoms.

Doctors are upholding the biomedical principles of medicine which incorporate a procedure by which patients are judged ill or well. This judgment occurs independently of the patient's own beliefs. Patients may be judged ill even though they may view themselves as well (screening asymptomatic patients) or judged to be well, even when they see themselves as ill (no biological explanation for the patient's symptoms).

In a 1975 study of inpatients in a general hospital, Lober [49] reported that *good patients* were described as trusting, coop-

erative, uncomplaining; and undemanding. Patients who interrupted a caregiver's established routine and created extra work were considered *difficult or problem patients*. But if such patients were perceived as being seriously ill, their demands were considered forgivable because the circumstances were beyond their control. If seriously ill patients were cheerful, cooperative, uncomplaining, and objective about their illness, they were regarded as *great patients*. Patients who were perceived as not being seriously ill but were demanding, emotional, and uncooperative were thoroughly condemned by caregivers, frequently discharged early, given sedatives, or referred to psychiatry. Thus, unless patients were seriously ill and/or *good patients*, they were labeled *difficult patients* and treated with less care.

Studies in primary care settings by Hahn [50] found that 15% of patients were viewed by doctors as difficult patients. These patients were more likely to have a psychiatric disorder, a functional impairment, greater health-care utilization and higher dissatisfaction with their care. This labeling occurs because patients have presented with "problems of living" rather than defined diseases in the hope of attaining the benefits of the sick role (exemption from social responsibility and relief of guilt). The doctor is reticent to exercise his or her unique social authority to grant these benefits and views such patients as difficult, because they do not fulfill the construct of the biomedical model.

The fibromyalgia patient is a "difficult patient"

Clearly patients with fibromyalgia have many of the characteristics of "difficult patients": a chronic illness without a readily identifiable lesion, multiple somatic complaints, a higher incidence of psychiatric disorders, greater health care utilization, and dissatisfaction with their care. Additionally, the advice of the doctor is often not followed, with patients turning to alternative sources of "medical expertise" often found in fibromyalgia networks around the country and the across the Internet. A patient with the new onset of fibromyalgia will initially attempt to fulfill the expectations of the sick role, while the disenchanted patient with long-standing fibromyalgia will often come to the doctor with a comprehensive belief system which in its range and power rivals the biomedical scientific belief system of the doctor. A search on *www.amazon.com* on May 3, 2002 returned 133 matches for books on fibromyalgia.

While on the surface the doctor-patient relationship is seemingly about common interests and goals, the doctor's view of the nature, cause, prognosis and therapy of the illness is increasingly countered by the patient's own explanations about the nature cause, prognosis and appropriate treatments for his or her illness [51].

Beyond the biomedical model

Describing the spaces occupied by disease, Foucault argued that diseases are conceptualized as primarily spatialized by their symptoms, secondarily spatialized by the presence of a discoverable pathologic lesion within the body, and tertiarily spatialized by the space they occupy in society [43]. It is the concept of this greater social space that diseases occupy that becomes especially relevant to chronic illnesses like fibromyalgia. This occurs because the presence of many biological causes of illness is strongly influenced, or even caused, by social factors. Illness is a multidimensional concept for which the cause of the biological lesion alone is not sufficient to explain all its features.

Illness, social integration and life events

In his classic 1897 treatise about suicide, Emile Durkheim [52] discussed how the risk of suicide varies with levels of social integration. Drawing on this hypothesis, researchers have explored the relationship between social support and illness [53]. Most of the research has focused on patients with unexplained medical symptoms and has consistently found higher levels of current and lifetime episodes of depressive disorders. Even in patients with well-defined chronic medical diseases, those with comorbid anxiety or depressive disorders have significantly more medical symptoms without identified pathology than do those patients without concurrent anxiety or depression. Additionally adverse childhood experiences and adverse events in adulthood like assault, domestic violence and natural diasters have all been associated with vulnerability to persistent syndromes like irritable bowel syndrome, interstitial cystitis and fibromyalgia [42].

Fibromyalgia and the role of gender

Fibromyalgia is more prevalent in women [12]. Explanations for this gender bias have revolved around both biologic and social explanations [54]. Studies of pain thresholds in pain-free, healthy persons have shown that, given the same intensity of stimulus, women report greater pain when compared with men [55]. The social explanation of an increased prevalence of illness in women (not just fibromyalgia) invokes the difference in the social position of women compared to men. Verbugge explored the higher rates of illness in women and amongst other factors found that women had greater illness rates because they were less involved in paid work and had stronger feelings of vulnerability to illness. Interestingly, when corrections were made for these factors, the male/female difference narrowed [56].

Fibromyalgia and labeling theory

A doctor's decision to interpret an individual's particular biologic state as being abnormal (diseased or ill) constitutes a label (diagnosis). In social theory, labeling has a negative connotation. Individuals deemed abnormal are labeled as such, and if this abnormality is also held to be socially abnormal, deviance results. Primary deviance refers to the actual defining of the biologic state as abnormal, while secondary deviance refers to the change in behavior of the patient that occurs as a consequence of labeling. Strong social pressures to conform to the label reinforce this behavior setting up a "self-fulfilling prophecy" [57].

When patients with widespread pain and multiple tender points are labeled as suffering from fibromyalgia, the subsequent change in behavior and the pressure to conform to the diagnosis create secondary deviance. This reaction on the patient's part arises from the social meaning and significance of the label the doctor has applied. When critics refer to fibromyalgia as a misleading and dangerous label, they are suggesting that the doctor is creating secondary deviance in the patient. The patient labeled with the diagnosis of fibromyalgia will fall into a self-fulfilling prophecy trap [3]. These critics suggest that it is the label that perpetuates the illness behavior, and not the underlying biologic state of the patient. By declaring "normal aches and pains" a disease, doctors are promoting the sick role and creating unnecessary somatization. Clearly, there are also patients who meet criteria for fibromyalgia but would prefer another diagnostic label because they want to avoid the stigmatization that might ensue from such a label. They may

prefer a label of a more "acceptable" (and thus less stigmatizing) disease like systemic lupus erythematosus or Lyme disease, even though close scrutiny would suggest that such patients would not meet definitions of these "adopted" diseases.

Others view fibromyalgia as a very useful label [58]. There is certainly a therapeutic effect of a diagnostic label. Patients are comforted when they can give their symptoms a name. This conferring of the sick role relieves them of personal responsibility for the cause of their symptoms. Researchers can also study this set of symptoms in a more uniform manner if formal criteria for diagnosis are created.

Is fibromyalgia a disease, a syndrome or an assortment of unexplained symptoms?

Before this question can be tackled, it is reasonable to examine the definition of disease itself. The traditional approach is to view disease as a biological problem. While this may seem logical, it immediately creates difficulties in explaining conditions (for which medical care is sought) without known biological mechanisms and involves the assumption that the line between physiology (normal) and pathology (abnormal) is well-demarcated. The latter phenomenon is especially apparent in diseases like diabetes, hypertension and hyperlipidemia.

The field of psychiatry has been especially subject to questions about its biological basis and the distinction between what constitutes normal and abnormal. Most psychiatric diagnoses do not have clearly defined biological mechanisms, and since the manifestations of mental illnesses are principally in altered behavior, which itself may be a consequence of labeling, critics like Thomas Szasz [59] have argued that psychiatrists do not identify real disease, they simply label inappropriate behavior and call it disease.

Another definition of disease is to break it down into its constituent parts. Dis-ease, would suggest that lack of feeling well (i.e. ill) is what constitutes disease. This concept of disease is unacceptable to doctors because it places the definition of disease firmly in the grasp of the patient and becomes synonymous with the lay concept of illness. Objectivity succumbs to subjectivity, and presymptomatic disease states cease to exist.

The view of disease as variance from what is socially ideal (or socially normal) suggests that variation from normal is only a disease if it creates social disadvantage. Thus Gilbert's disease is not a disease, just a biological variant. This view suggests that extremes of continuously distributed variables are only diseases when social disadvantage is created. It also accommodates for differences, in what may be considered a disease, based upon the established social norms of communities and the limits of social acceptability. Foucault has traced the historical change in the acceptance of insanity from a part of everyday life to a threat to society, illustrating the dynamic nature of the definition of insanity [44]. Osteoarthritis and the loss of agility with age may be viewed as a normal phenomenon or a disease depending on the social norms of the community.

Fibromyalgia has mostly been referred to as a symptom-complex or a syndrome, with most investigators being quite emphatic that it is not a disease [13;58], i.e. it does not have a classic biomedical basis. On the other hand, if fibromyalgia is considered to be disease based on the social construct, the disease-syndrome argument becomes irrelevant. The most ardent sup-

porters of fibromyalgia as a disease are patients with this disorder. It is not uncommon for sociopolitical pressures to coerce biomedical acceptance of nonspecific syndromes, even to the extent of renaming them, as in the example of chronic fatigue syndrome being renamed postviral syndrome.

When do symptom-complexes become a disease? This is clearly a complicated issue that not only depends on the discovery of a reductionistic biologically plausible explanation, but also on sociopolitical forces and changing sociocultural norms [60].

Reconstructing the biomedical model

Patients seek medical consultation for symptoms. Doctors diagnose diseases to explain the symptoms. An impasse occurs when no pathologically defined disease can be found to explain the symptoms. The patient does not fit the biomedical model of disease and alternative explanations are suggested, most of which invoke a psychiatric disorder. Most patients are unwilling to accept the stigma of a psychiatric label. This results in secondary deviance (the social effects of negative labeling) which may cause the patient to "doctor shop" or adopt a more acceptable biomedical label like systemic lupus or Lyme disease. Furthermore, the negative effects of labeling may create a self-sustaining loop of worsening symptoms and increasing dissatisfaction with care. Sociopolitical forces like advocacy groups may force acceptance of certain syndrome clusters as bona fide diseases.

Up to one third of all symptoms in primary care settings are unexplained [61]. This failure to address "unexplained" symptoms is an important limitation of the biomedical model of disease. Prior to the advent of psychoanalysis, unexplained symptoms were considered functional disturbances of the brain. Subsequently, unexplained symptoms became the province of psychiatry and somatization was proposed as the mechanism by which mental disorders became physical symptoms. Sharpe has suggested that we revert to the pre-psychoanalytic era by approaching unexplained symptoms as reversible disorders of brain function rather than as a fixed pathology of a purely psychological nature [62]. Since patients still prefer to address unexplained symptoms with their generalists and only a minority accepts referral to psychiatry, Sharpe has suggested a "psychologically augmented medical consultation" as a strategy to improve patient outcomes.

As committed students of science, doctors uphold the intrinsic validity of the biomedical model and view the constructs of biomedicine as pure science, impervious to social forces. There are two challenges to this view. The first challenge is the concept expounded by Kuhn, that growth in science can be viewed as a succession of successful puzzle solutions utilizing practices that are deeply anchored in paradigmic practices. "A paradigm is an exemplary piece of scientific work which creates a research tradition within some specialized area of scientific activity" [63]. A paradigm is a working model upon which further research is based, creating a family of related projects dedicated to solving puzzles. When a particular puzzle is insolvable, a new model may be generated in order to solve the puzzle. Once solved, a new paradigm that accommodates the anomaly is established. Thus, scientific growth is not a journey towards an infinite absolute truth but a series of temporarily valid solutions (often determined by prevailing views) that provide successively

better explanations. The scientist does not function in an atomistic, individualistic fashion, but rather as one of many scientists working to validate an established paradigm while willing to accept a newer, better paradigm when appropriate.

The second challenge to the invulnerability of science comes from a greater philosophical concept which espouses that all reality is socially constructed [64]. What is "real" to the patient and the "knowledge" they possess about the certainty of this reality, is not qualitatively different from what is real to the scientific community. Both sets of reality occur within specific social contexts and questions of mutually exclusive validity are immaterial.

Neurasthenia and chronic fatigue syndrome: a biosocial model

The constructivist view states that we should accept the vision that diseases exist not just in the body but in a greater social space and that the constructs of medicine must encompass the "whole" patient, a concept that places the patient (and thus their diseases) in the context of their place within society. We should also accept that our scientific views are often shaped by prevailing social thought. The latter point is extremely well-illustrated by Abbey's comparison of neurasthenia and chronic fatigue syndrome, two virtually identical diseases described greater than a century apart [2].

When George Beard described neurasthenia in 1869, explanations for this disease focused on the major themes of scientific interest during the late 19th century: electricity (Edison), conservation of energy (Helmholtz), and evolution (Darwin). Neurasthenia was viewed as a consumption of nervous energy (electricity), of which there was a finite amount (first law of thermodynamics). This over consumption of energy was a consequence of the demands and stresses of modern times which reflected the pressures (evolutionary) of a capitalist society.

Explanations regarding the cause of chronic fatigue syndrome reflect the prevailing views of the 1990's: which have focused on the immune system, infections and environmental toxins. Thus, chronic Ebstein-Barr virus infection, multiple chemical sensitivity syndrome and systemic candidiasis have all been elaborated as explanations for chronic fatigue syndrome.

Conclusions

The controversy surrounding fibromyalgia reveals two major themes. There are investigators (and patients) who are committed to establishing credentials for fibromyalgia as a bona fide disease by focusing on traditional pathophysiologic mechanisms. Most of these currently involve unraveling mechanisms of pain perception and HPA axis disorders. Non-traditional etiologic factors like stress and a history of childhood or adult trauma are considered as either yet-to-acquire known pathophysiologic mechanisms or thought to function as disease co-factors, lowering thresholds and causing somatosensory amplification of symptoms. The negative label of "non-disease" applied to patients with fibromyalgia is perhaps the cause and not the result of the maladaptive coping strategies observed in these patients.

The second theme is a sociological and philosophical one that suggests that the narrow constructs of biomedicine exclude symptom-based diagnoses like fibromyalgia and insinuates that physicians and patients have parallel disease constructs each with their own set of views regarding dis-

ease etiology, pathogenesis and treatment. This theme emphasizes the social role of the doctor as he or she is constantly upholding social and moral norms of society by controlling patient access to the "sick role". This theme also suggests that a disease occupies not just the physical space of the body but the wider social space occupied by the diseased individual.

Fibromyalgia as a disease construct will undergo one of two fates. It may remain a biomedical puzzle that will eventually be solved by a new biomedical paradigm that will discover the elusive pathologic lesion. Alternatively, our current concepts of disease will broaden to accommodate medically unexplained symptom complexes like fibromyalgia.

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