

The Changing Nature of Fibromyalgia

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Key Points

The definition and content of fibromyalgia changed substantially over time, as did the patients with the disorder.

Tender points and widespread pain were key features of the 1990 criteria.

Tender points were abandoned and symptoms, including somatic symptom reporting and cognitive problems (Fibro Fog”), were added to criteria in 2010.

Fibromyalgia and 19th century neurasthenia are often indistinguishable.

Fibromyalgia does not fit the definition of a categorical disorder, but represents the end point of a continuum of polysymptomatic distress.

Abstract

Fibromyalgia is a common but contested illness. Its definition and content has changed repeatedly in the 110 years of its existence and in the 60 years since it became a generalized pain disorder. The most important change was the requirement for multiple tender points and extensive pain that arose in the 1980s, features that were not required previously. By 2010, a second shift occurred that excluded tender points, allowed less extensive pain, and placed new reliance on symptoms that included increased somatic symptoms reporting and cognitive difficulty (“Fibro fog) that had never been part of past definitions or content. Evidence shows that fibromyalgia is not a categorical disorder, but rather a dimensional condition that represents the end of a spectrum of polysymptomatic distress. Fibromyalgia is closely allied and often indistinguishable from neurasthenia, a disorder of the late 19th and early 20th century that lost favor when it was perceived as being a psychological illness. Fibromyalgia is also associated with psychological illness and socio-demographic disadvantage. However, its status as a “real disease,” rather than a psychocultural illness, is buttressed by social forces that include support from official criteria, patient and professional organizations, Pharma, disability access, and the legal and academic community.

Main Text

In the 110 years of its existence fibromyalgia evolved from a regional pain disorder to a multiple symptom disorder that was indistinguishable in most respects from neurasthenia, a disorder of the 19th century that was abandoned in the 1930s with the recognition that it was a psychological illness. In its evolution, fibromyalgia changed course several times

(Table 1). The most important change was the mandatory requirement of tender points and generalized pain. Fibromyalgia became officially established by formal criteria in 1990,¹ after which a multiplicity of symptoms became a central component of the illness. By 2010 it could be diagnosed by symptoms and could be seen as the end of a continuum of polysymptomatic distress.² Fibromyalgia is comorbid with a series of other somatic symptom disorders, is associated with psychological illness, and is strongly influenced by social forces. In this paper we examine the history and issues in its transformation. Pain and symptoms, even in psychocultural disorders, must be expressed through neurobiologic mechanisms, which are only touched on briefly here, but will be the subject of a future article

The fibromyalgia controversy

Fibromyalgia is a contested illness,³ a type of illness “where sufferers claim to have a specific disease that many physicians do not recognize or acknowledge as distinctly medical.”⁴ The underlying controversy is about whether the disorder is “real,” not in one’s head, not psychosomatic; and not primarily a social construction or psychocultural disorder.^{5,6} A psychocultural disorder is one that is shaped primarily by psychological factors and societal influences, although other factors can also contribute. The primary contemporary dispute about fibromyalgia is whether psychocultural factors explain fibromyalgia content or whether fibromyalgia is largely a product of disordered central pain processing.⁷

The evidence for a psychocultural disorder is strong. The neurobiologic etiology is supported primarily by imaging and neurophysiologic associations.⁸ However, there is as yet no compelling evidence that an underlying CNS disturbance contributes *in a substantial or clinically meaningful* way to the fibromyalgia phenotype. In addition, neurobiologic associations depend on fibromyalgia being a discrete entity, for which there is no evidence. Even if fibromyalgia is considered to be largely a psychocultural disorder, a strong body of criticism suggests that the definition and content of fibromyalgia is arbitrary, and the methods of assessment illusory.^{6,9-16}

A brief history of fibromyalgia

The start of fibrositis

While fibromyalgia is contested, it is still a common diagnosis that is recognized, if not always approved of, by physicians, patients, governments, pension and disability systems, scientific organizations, and the diagnostic codes of the International Classification of Diseases (ICD).¹⁷ Fibromyalgia was known as fibrositis until 1990 when the American College of Rheumatology criteria recommended changing the name to fibromyalgia,¹ a name that had been proposed by Hench¹⁸ and championed by Yunus.¹⁹ Fibrositis arose as a term to describe painful local or regional areas. In 1903, Gowers wrote, “I think we need a designation for inflammation of the fibrous tissue.... We may conveniently follow the example of ‘cellulitis,’ and term it fibrositis.”²⁰ By the time the term fibrositis had run its course and was discarded, virtually every organ system and body region had been

identified as a site for fibrositis, including the eye, throat, breast, testicle, liver, pancreas, and on and on.²¹ The idea of sterile inflammation and specific anatomic abnormalities, espoused by Gowers and others,²⁰⁻²⁵ was generally abandoned by the 1950s. Pain and stiffness were the major symptoms of this illness. For many, fibrositis was, not surprisingly, a “wastebasket” diagnosis.⁹

Generalized fibrositis

Gower’s description had set the stage for generalized fibrositis. If fibrositis could occur in almost any region, why could it not occur in many regions simultaneously? In a comprehensive review of fibrositis in 1951,²⁶ Freyberg, one of the founders and early leaders of the rheumatology movement, divided fibromyalgia into generalized fibrositis and localized fibrositis. He warned of “psychogenic rheumatism that might be difficult to distinguish from fibrositis.” In Freyberg’s description of fibrositis he noted that patients could become tired because of discomfort and fatigue; there might be “little or no tenderness, but at sites of greater trouble there may be moderate, generalized tenderness. Dampness, cold, rain and snow usually worsen [and] warmth, dryness and high barometric pressure characteristically relieve” the symptoms of fibrositis.²⁶ Similar description were provided by Graham (1952)²⁷ and Traut.²⁸ Traut, in 1968, provided a description of “generalized” fibrositis that included “generalized deep tenderness,” generalized aching and stiffness, anxiety, and headaches.²⁸ Patients “often [had] a history of colitis.” They were “always tired” and were “poor sleepers.”

Smythe, whose criteria led to the expansion and acceptance of fibrositis, described fibrositis pain in detail, but wrote only of stiffness and “an unusual degree of tiredness and fatigue” as symptoms of fibromyalgia in the 1966 edition of the standard rheumatology textbook.²⁹ Six years later (1972), in the next addition of the textbook,³⁰ he reported “complaints of “chronic fatigue, and that patients “all sleep badly, with marked morning stiffness.” As with preceding authors, there was little description and interest in other symptoms.

Unrefreshed sleep and tender point criteria

Smythe’s colleague, the Canadian psychiatrist Moldofsky, reported an abnormality of Stage 4 sleep in fibrositis patients in 1975 and proposed that the “fibrositis” symptom complex be considered a “non-restorative sleep syndrome”.³¹ A year later he reported that “the emergence of musculoskeletal symptoms and increase in muscle tenderness is induced by a disturbance of non-REM sleep in otherwise young, healthy subjects” and “we were able to artificially induce in our healthy subjects not only the symptoms found in our ‘Fibrositis’ patients, but also a similar physiologic non-REM sleep disturbance.”³² Here was the first of a series of proposed mechanisms for fibrositis, and a step toward scientific legitimacy.

Moldofsky and Smythe (1976) went on to publish, “Two contributions to the understanding of the ‘fibrositis’ syndrome,”³³ in which they proposed a scientific mechanism for fibrositis as well as the first-ever usable set of criteria for diagnosis. The authors proposed serious, reasonable criteria for diagnosis that appeared to have a scientific basis: “widespread

aching for longer than three months, disturbed sleep with morning fatigue and stiffness,” and tenderness in at least 11 of 14 pre-specified tender point sites. With reproductions of the sleep electroencephalograms and specification of tender sites in the article, fibrositis gained substantially in scientific credibility and piqued interest in the syndrome.

In the decade before the adoption of the 1990 American College of Rheumatology (ACR) criteria for fibromyalgia– which would become the *de facto* official definition, two ideas about criteria contested: limited pain sites and tender points, but with multiple symptoms versus generalized pain, a large number of tender points, and few symptoms. Yunus (1981) proposed criteria that were mainly concerned with symptoms or their modulation by external factors.¹⁹ There needed to be generalized musculoskeletal aching at 3 or more sites, plus 3 of the following ten features: chronic anxiety or tension, fatigue, poor sleep, chronic headaches, irritable bowel syndrome, subjective soft tissue swelling, numbness, pain modulation by physical activities, pain modulation by weather factors, pain modulation by anxiety/stress. “Four tender points will also satisfy the criteria if the patients has 5 of the above 10 features. The criteria operationalized and expanded the Freyberg-Graham-Traut descriptions. At the same time, other authors assembled criteria that resembled the Smythe and Moldofsky criteria, differing primarily in the number and location of the required tender points.³⁴⁻³⁶ Although Yunus called his symptoms “minor criteria,” they took on particular importance because it was quite easy to satisfy aching in 3 sites and having 4 or 5 tender points. In addition, the Yunus minor criteria suggested a psychosomatic illness.

The 1990 ACR criteria

In 1990, the contending definitions were examined in a criteria study that produced the “official” ACR criteria: 11 of 18 tender points and the presence of widespread pain for diagnosis.¹ No other symptoms were required. The 1990 criteria marked the start and growth of clinical era of fibromyalgia (Figure 1) by providing a powerful endorsement from a respected scientific body as well as a scientific study, though not all agreed that the fibromyalgia idea of criteria made sense.^{6, 9-16} Figure 1 suggests that fibrositis had little public impact before the 1990 criteria.

In the period between 1990 and 2010 scientific data began to come in that linked fibromyalgia to neurobiologic findings. The interest sparked by the ACR criteria led to numerous studies that identified many more symptoms and found co-diagnoses with illnesses such a chronic fatigue and irritable bowel syndrome. Evidence of psychosocial disadvantage and psychological illness accumulated.

The ACR 2010 revision

In 2010 a revised definition of fibromyalgia came into being in the form of *The American College of Rheumatology Preliminary Diagnostic Criteria for Fibromyalgia and Measurement of Symptom Severity*.² These criteria dropped the tender point requirement and replaced it with self-reported symptoms. A diagnosis now required many self-reported painful body regions and high levels of somatic symptoms, fatigue, unrefreshed sleep, and cognitive

problems. The changes in the definition of fibromyalgia which began with Gowers' fibrositis, ended in a symptom disorder in which pain was only one, if not the most prominent, of many symptoms. Outside the rheumatology community fibromyalgia was considered differently. It was seen as primarily as one of a series of somatic symptom disorders; and evidence continued to accumulate that it represented the end of a spectrum of polysymptomatic distress.

Psychogenic rheumatism and psychological illness

The *bête noire* of fibromyalgia is psychological illness. There is reason for lay and medical suspicion of the symptom complaints of those with fibromyalgia: there are too many symptoms, and the symptoms appear too severe and too unusual, the patients too healthy. There is an increased lifetime prevalence of many DSM AXIS I and II and other psychological disorders.^{37, 38} In addition, for almost any symptom characteristic or comorbid illness fibromyalgia patients with have more abnormal scores compared with control groups. Only patients with end stage renal failure have lower quality of life scores.³⁹ Fibromyalgia patients are those that physicians don't want to see.⁴⁰ They are "heartsink" patients⁴¹ who fit the description of Leap, "Men and women whose presentations render useless the DSM guidelines..."⁴² "We think they are all crazy," a distinguished rheumatology division head and Professor of Medicine told us, reflecting a not uncommon view. The medical and lay literature contains many descriptions of the antagonism between physicians and "difficult" patients with fibromyalgia. Difficult patients are characterized by "psychosomatic symptoms, at least mild personality disorder, and Axis I (major) psychopathology, and most had more than one of these characteristics."⁴³

Psychogenic rheumatism

Psychogenic rheumatism, if imprecise, was a well-understood and diagnostically important term in rheumatic diseases in the four or five decades beginning with the 1930s. The specific phrase appeared first around 1939,⁴⁴; however, the idea of a psychological etiology or intensification of rheumatic symptoms was common throughout the 19th and 20th centuries "Primary fibrositis is the principal rheumatic condition from which psychogenic rheumatism must be distinguished," wrote Boland in 1947.⁴⁵ The Glasgow psychiatrist Halliday wrote that "the various terms which indicate 'non-arthritis' frequently cover anxiety states or hysteria ..."^{46, 47} Freyberg stated that psychogenic rheumatism was the commonest form of non-articular rheumatism. Boland, who also provided a series of "for example" definitions in a large table, considered that psychogenic rheumatism was a convenient and compact term.⁴⁵ Reynolds provided a review and definition of psychogenic rheumatism 1978.⁴⁸ Reynolds was not a believer in fibromyalgia. For Reynolds fibrositis was psychogenic rheumatism.

Still, one really could not define what psychogenic rheumatism meant in a way that was valid and reliable, and the term was demeaning. Bennett, one of the founders of the fibromyalgia movement summed up the modern view, "Except for describing a very small minority of patients with bizarre pain, the term 'psychogenic rheumatism' has no place in contemporary medicine."⁴⁹ Psychogenic rheumatism would also disappear because the old

meaningless word, rheumatism was a relic of another time. But what psychogenic rheumatism represented in patients with non-articular rheumatism in the first three-quarters of the 20th century would be allowed by the new definitions of fibrositis and fibromyalgia that emerged with the 1990 and 2010 criteria. If there was no conscious attempt to remove psychogenic rheumatism, whatever it was, then fibromyalgia could provide a convenient home (diagnosis) for many with psychological problems.

Fibromyalgia and neurasthenia

Fibromyalgia became a popular illness, but it is uncertain how many people have the disorder. Ehrlich points out that “No one has FM until it is diagnosed. FM is an iatrogenic syndrome because it has to be named by a doctor to exist.”¹⁵ Thus, there remains a difference between the number of people who satisfy criteria for fibromyalgia and the number who actually seek care for symptoms that are cast by physicians or patients as fibromyalgia. The estimated (potential) prevalence of fibromyalgia is around 2-4% worldwide.⁵⁰ The growth of fibromyalgia can be investigated by using a simple Google tool, Ngram,^{51, 52} to graph the number of time the words “fibrositis, fibromyalgia and neurasthenia” have been used in books, adjusted for the number of books available (Figure 1). Fibrositis arises around 1940, but decreases somewhat thereafter. Fibromyalgia becomes the dominant term after 1990, an effect of the 1990 ACR criteria. By contrast, neurasthenia, whose symptoms are very similar to fibromyalgia, is much more popular overall, but dies out as the 20th century progresses. In 2012, a search of the US Amazon book site identified 1,894 fibromyalgia titles currently available for sale, while a search of English books in Google Books found approximately 293,000 titles (hits) containing “fibromyalgia” overall and 26,400 with dates in 2011.

If, as shown in Figure 1, fibromyalgia is a relatively new disorder, was there no such illness prior to 1940 or were the symptoms of fibromyalgia present earlier, but differently named, perhaps as neurasthenia? Shorter points out that each era has its own illnesses, so it is not impossible that fibromyalgia is a new disorder. “As doctors’ own ideas about what constitutes “real” disease change from time to time due to theory and practice,” he writes, “the symptoms that patients present will change as well.”⁵³ But it might also be that fibromyalgia is a disorder based on a rearrangement, revaluation and redefinition of symptoms that were always present in what Shorter calls the “symptom pool.”⁵³

The prevalence of neurasthenia in its heyday is not known, as there were no population-based data at the time of its ascendancy. However, neurasthenia accounted for 6-11% of total discharges from the late 1890s to 1930 at the National Hospital, Queen Square, London, after which it virtually disappeared.⁵⁴ Taylor indicates that “Neurologists, not psychiatrists, continued to see the disorder well into the 20th century. Neurasthenia did not disappear, but was reclassified into psychological diagnoses.”⁵⁴ Wessely observed it this way, “From one of the most frequently diagnosed conditions in medical practice, neurasthenia disappeared almost as rapidly as it appeared.” See Figure 1. “It was clear that neurasthenia was shifting from being the concern of neurology to psychiatry. This change was of critical importance, since once neurasthenia was viewed as psychiatric, a principal social function was lost.”⁵⁵

What was neurasthenia?

Neurasthenia was first proposed by Beard in the US in 1869 as, at a first glance, a disorder of physical and mental fatigue.⁵⁶ Wessely reports, “Mental fatigue was an integral feature of neurasthenia. Kraepelin (1902) wrote that 'the accustomed work is carried out with increasing difficulty, requiring greater exertion and more frequent rests. They are easily distracted by little things and are inattentive. Twice the usual time is spent in reading the paper... they are forgetful with names and figures... They assert that the memory is becoming profoundly affected, and that the judgment is failing.' Thus described, mental fatigue resembles “fibro fog.” Fibro fog is new to fibromyalgia. Prior to 1990 only one “hit” is noted for fibro fog in a Google search, and the decade of the 1990s produced 104 hits. However the next 10 years added 13,800 results. Fibro fog, relabeled as cognitive difficulties, was incorporated into the 2010 ACR fibromyalgia criteria along with overall fatigue.

In Wessely's classic essay on neurasthenia, he describes what we might identify as *all of the other symptoms of fibromyalgia*: “‘Sufferers from neurasthenia often time wonder and complain that they have so many symptoms; that their pain and distress attack so many parts and organs' (Beard, 1880). Beard listed over 70, with special attention being paid to specific areas: cardiac, gastrointestinal, temperature regulation, paraesthesiae and pain syndromes. Oppenheim (1908) wrote that 'the symptoms of neurasthenia are so numerous that it is impossible to describe them in detail', but then devoted 17 pages to such a description.”⁵⁵ A person with neurasthenia today would most likely satisfy fibromyalgia criteria and be welcomed into that diagnosis. The very strong resemblance of fibromyalgia to neurasthenia is a key observation. Time brings clarity to confusing illnesses of the past, and we now recognize that hysteria, neurasthenia, and railway spine^{13, 57, 58} were almost always psychogenic disorders.

Why fibromyalgia succeeded

The primary requirement for the success of psycho-cultural illness is that it must not be perceived as being psychological (not real). Disorders that are primarily psychogenic attract societal attention and disapprobation, particularly when they ask for social advantage or disability pensions. The rise of fibromyalgia and the disputes it has engendered represents the age-old battle over psychogenicity. All other things being equal, fibromyalgia should have failed. It began as a simple local pain disorder, but evolved over time into one that had multiple somatic symptoms and features that many considered psychosomatic. If these features were the death knell of neurasthenia, they should have also spelled the death of fibromyalgia. But they didn't. The era was different, and powerful cultural forces stood behind fibromyalgia and fought against the idea of a psychogenic illness.

The most important factor in establishing fibromyalgia was its endorsement by a respected medical society of scientists and clinicians, the American College of Rheumatology in 1990, and figure 1 demonstrates that the increase in fibromyalgia words began just after 1990.

Patient organizations also played an important role. For patients, a scientifically valid fibromyalgia provided legitimation of symptoms and entry into acceptable diagnosis.⁶ Patients groups sprung-up, organized, published journals, and spread worldwide as the Internet expanded.^{59, 60} They lobbied the US congress, state legislatures and the National Institutes of Health (NIH), and their persistence was an important force in persuading a doubting NIH that funds should go to fibromyalgia research. Between 1996-1999 Social Security disability became available.⁶¹ Specifically, fibromyalgia was designated as a “medically determinable physical impairment” by the presence of tender points. Railway spine reemerged when cases were made that trauma, any trauma, could cause fibromyalgia.^{62, 63} A huge legal industry developed to serve persons injured who then developed fibromyalgia. In 2012 the search term “fibromyalgia AND lawyer produce 1.3 million hits.

As fibromyalgia became a legitimate illness, it also became an attraction for academia in terms of funding and publications. The astounding annual growth in publications about fibrositis and fibromyalgia is shown in Figure 2. The pharmaceutical industry began research into fibromyalgia treatment in the 1990s, and pregabalin (Lyrica) was approved by the US Food and Drug Administration (FDA) in 2007, followed soon after by approval of other fibromyalgia drugs. Figure 2 suggests that an approximate 100% increase in publications occurred starting in 2006. The pharmaceutical industry engaged in extensive direct to patient (DTP) advertising as well.³ The well-dressed middle class and above patients with fibromyalgia seen in the advertisements all seemed to benefit from the treatments which, in real life, were minimally effective at best.^{64, 65} The message concerning “overactive nerves” reinforced the “real disease” message. Pharma actively funded physician education and patient support groups. There was hardly any prominent fibromyalgia-supporting physician who did not receive financial support from Pharma in the last decade. The effect of these societal influences was to provide a counterweight to the fibromyalgia as a psychological illness concern — enough doubt for fibromyalgia to remain viable, even if contested.

Somatic symptoms and continuum of polysymptomatic distress

In agreement with many, Fink and Schröder characterized fibromyalgia as one of many Bodily Distress Syndromes,⁶⁶ in which psychological abnormality might not be a necessary concomitant. “Somatization is an extremely frequent phenomenon and basic manifestation of human life. The development of somatization-related disorders is only the tip of the iceberg.”⁶⁷ The idea that fibromyalgia might be part of a continuum, a dimensional disorder rather than a categorical one, surfaced soon after tender point criteria were developed.⁶⁸ Following the establishment of the 1990 ACR criteria for fibromyalgia, epidemiological studies addressed this point directly. In a study of persons in the community who satisfied the widespread pain criterion, Croft et. al wrote in 1994, “Fibromyalgia does not seem to be a distinct entity in the general population,”⁶⁹ and two years later put the question: “More pain, more tender points: is fibromyalgia just one end of a continuous spectrum?”⁷⁰ In a study of clinical data, Wolfe concluded that “Tender points are linearly related to fibromyalgia variables and distress, and there is no discrete enhancement or perturbation of fibromyalgia or distress variables associated with very high levels of tender points.”⁷¹

In a comprehensive review of fibromyalgia, Wessely wrote that he was “unaware of any study” that supported the view that fibromyalgia was a categorical disorder.⁷² “Most thinking on fibromyalgia follows a [categorical] model,” he stated, “but we favor a [dimensional] view,” and suggested that fibromyalgia (fatigue and myalgia syndromes) “are arbitrarily created syndromes that lie at the extreme end of the spectrum of polysymptomatic distress. Definitive evidence to support or refute this view will come from primary care or community samples, not the study of specialist populations. A study that takes the extreme end of the spectrum, represented by selected samples of patients referred to rheumatology or pain services, and compares them with non-fatigued controls, will produce a ... categorical solution but for spurious reasons.” With the publication of the 2010 ACR criteria that was modified for survey research,⁷³ and in an earlier predecessor survey method⁷⁴, it became feasible for to address these questions in the general population for the first time, with results that confirmed the continuum hypothesis.

Evidence for a dimensional disorder

Given a series of relevant predictors, Kessler indicated that the preferences for dimensional assessments is “whether the predictors are consistently related to differences in symptom severity across the full relevant range of the dimensional distribution. If they are, then analysis of the dimensional version of the symptom scale makes most sense”⁷⁵ “The spectrum of distress reflects the human condition.”⁷⁶ Polysymptomatic distress refers to the common set of symptoms from the human “symptom pool”.⁵³ Practically, one can think of the polysymptomatic distress spectrum as representing multiple different symptoms, each potentially at a different level of severity (e.g., none, mild, moderate, severe). The polysymptomatic distress scale of the 2010 ACR fibromyalgia criteria survey modification represents the sum of the widespread pain index and the symptom severity scale, and is highly correlated with fibromyalgia symptoms.² To examine Wessely’s end of spectrum idea in a community sample, we used data from 2,322 randomly selected subjects in a 2008 German population study.⁷⁷[FW et al submitted] Use of the survey criteria allowed us to examine symptoms in randomly selected persons in the community. Figure 3, left shows the distribution of the Patient Health Questionnaire-15,⁷⁸ a measure of somatic symptom severity. The mean PHQ-15 score of patients with fibromyalgia is 11.3, and occurs at the 96th percentile of PHQ-15 scores. The mean value of the polysymptomatic distress scale in subjects with fibromyalgia is 18.6, a value that occurs at the 98th percentile of polysymptomatic distress scores. The curves of figure 3 also mirror data on tender points.⁶⁸ There is little doubt the fibromyalgia occupies “the extreme end of the spectrum of polysymptomatic distress.”⁷²

Are “predictors consistently related to differences in symptom severity across the full relevant range of the dimensional distribution?” We evaluated the relation of PSD to the SF-36 physical and mental component summary scores (PCS and MCS), PHQ-15 somatic severity index and PHQ-9 depression index in the German population survey (Figure 4). Graphical and statistical tests showed no evidence of non-linearity in the PSD relationships, and there was substantial correlation between the PSD and PHQ-15 ($r=0.754$), PHQ-9 ($r=0.614$), PCS ($r=-0.629$) and MCS ($r=-0.531$). Graphs of the same variables vs. the

probability of fibromyalgia show similar relationships. Thus all data offer no support for the categorical hypothesis and substantial support for the continuum hypothesis.

Summary

The definition of fibromyalgia has changed considerably over time. Fibromyalgia seems to be a somatic symptom disorder with remarkable similarities to neurasthenia. It represents the end position on a continuum of distress. While psychological issues are clear, powerful societal forces are marshaled on behalf of fibromyalgia, and it seems likely that they will sustain the fibromyalgia, at least for the present. Studies of neurobiologic mechanisms need to consider the dimensional nature of the disorder and its variability.

Table 1. Some landmarks in the evolution of fibromyalgia

Illness/Author	Time period	Characteristics
Neurasthenia	1868-1930	Similar to current fibromyalgia. Pain, mental fatigue, myriad symptoms
Fibrositis (Gowers)	1903	Primarily local, with local symptoms including pain & stiffness
Freyberg	1951	Predominantly generalized,
Graham	1953	Pain, stiffness, and soreness
Traut	1965-1968	Fatigue, poor sleep, headache, colitis, tender points, anxiety
Smythe- Moldofsky	~1976	Sleep is causal. Tender point counts are diagnostic. Widespread aching, fatigue, very high TP count (11/14) (79%) Requirement of specific TP count & generalized or widespread pain
Yunus	1981	Symptoms important. TP could be few ~5 TP (<25%), pain or aching in only three areas
Goldenberg, Bennett, Wolfe	1980-1989	Similar to Smythe-Moldofsky, but ~60% TPs are positive
Wolfe et. al (ACR 1990 Criteria)	1990	TP11/18, widespread pain (61%)
Wolfe et. al (ACR 2010 criteria)	2010	Many painful body regions and high levels of somatic symptoms, fatigue, unrefreshed sleep, and cognitive problems. Can be seen as a continuum

Google books Ngram Viewer

Graph these **case-sensitive** comma-separated phrases:
between and from the corpus with smoothing of .

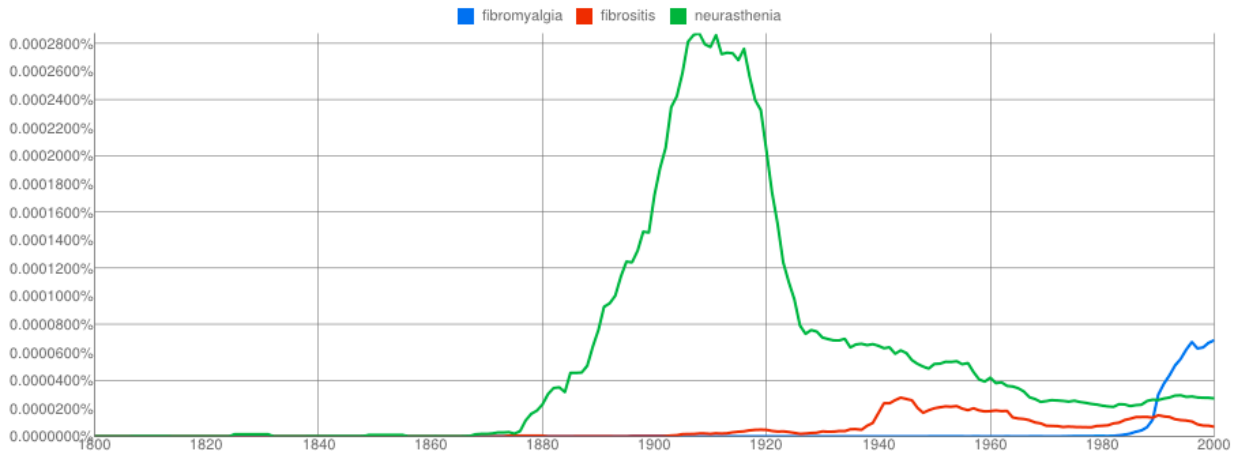


Figure 1. The rise and fall of neurasthenia, the beginning of fibrositis, and the growth of fibromyalgia following the 1990 ACR criteria based on word searches in Google.

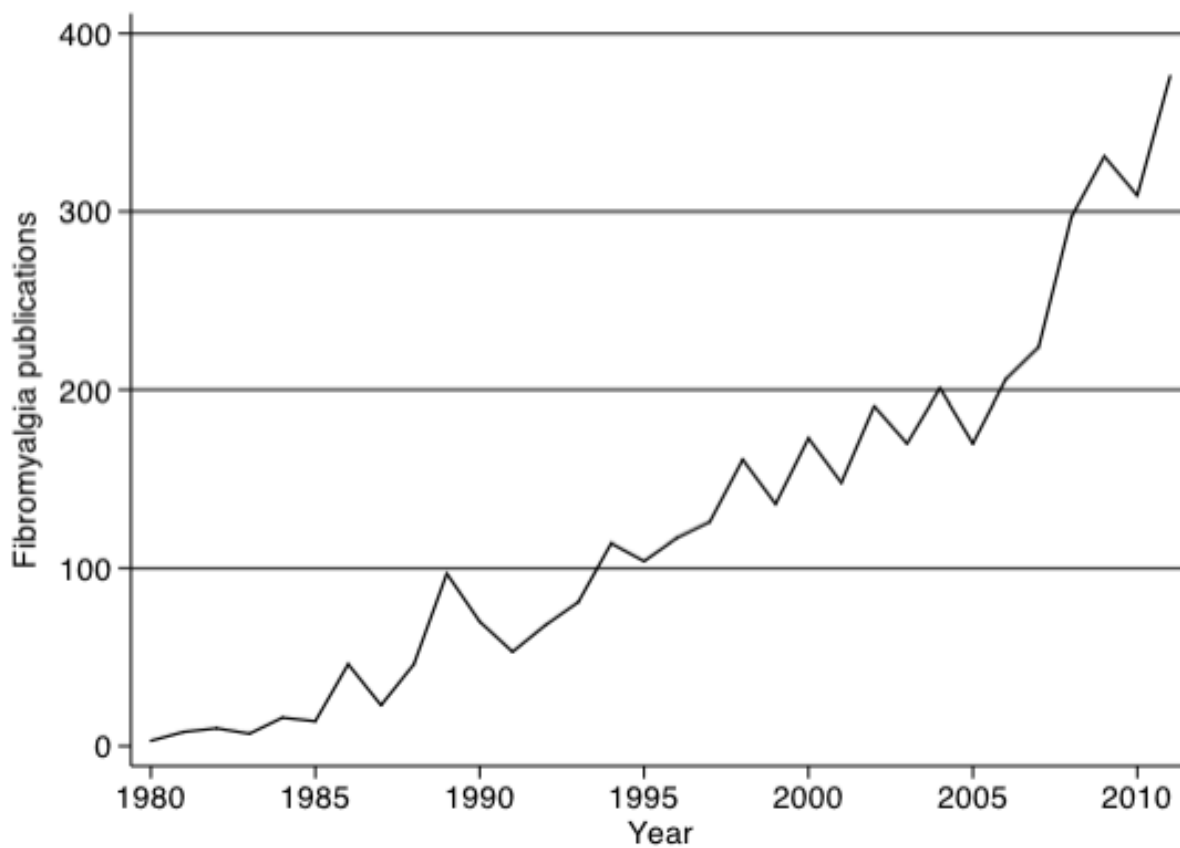


Figure 2. Annual scientific publications on fibrositis or fibromyalgia as identified in Pub Med.

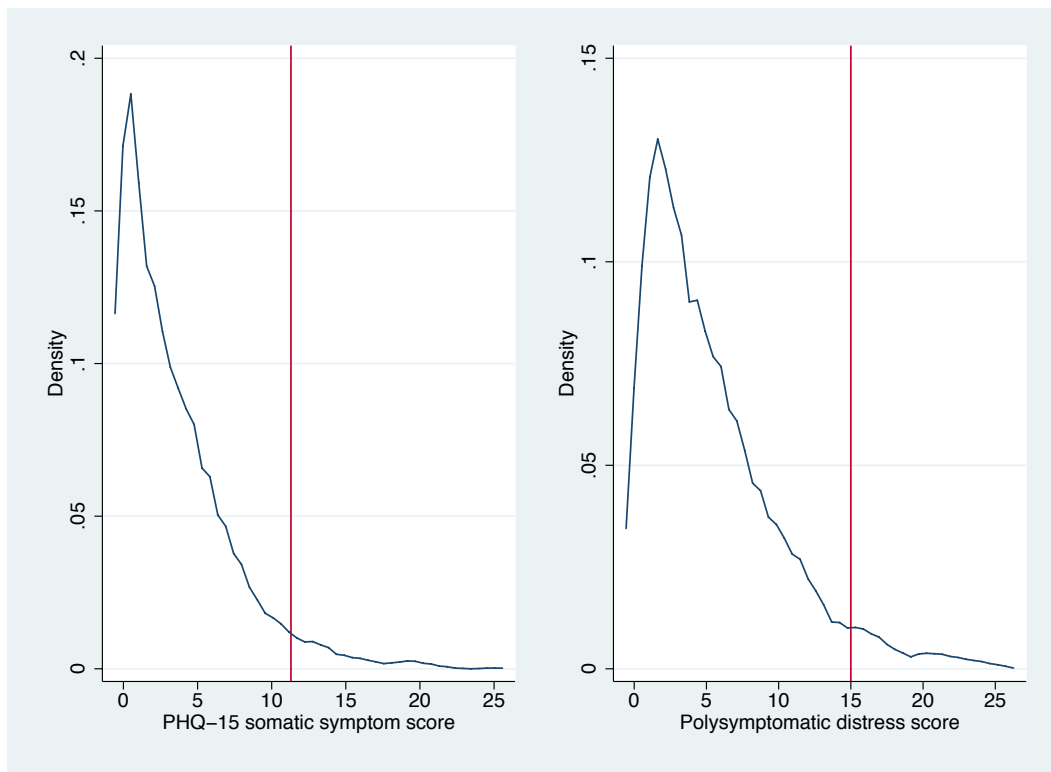


Figure 3. The distribution of the Patient Health Questionnaire-15, a measure of somatic symptom severity in the general population (left). The mean PHQ-15 score of subjects with fibromyalgia is 11.3, and occurs at the 96th percentile of PHQ-15 scores. The mean value of the polysymptomatic distress scale (right) in subjects with fibromyalgia is 18.6, a value that occurs at the 98th percentile of polysymptomatic distress scores. The vertical line at 15 indicates the value that best separates fibromyalgia from non-fibromyalgia subjects.

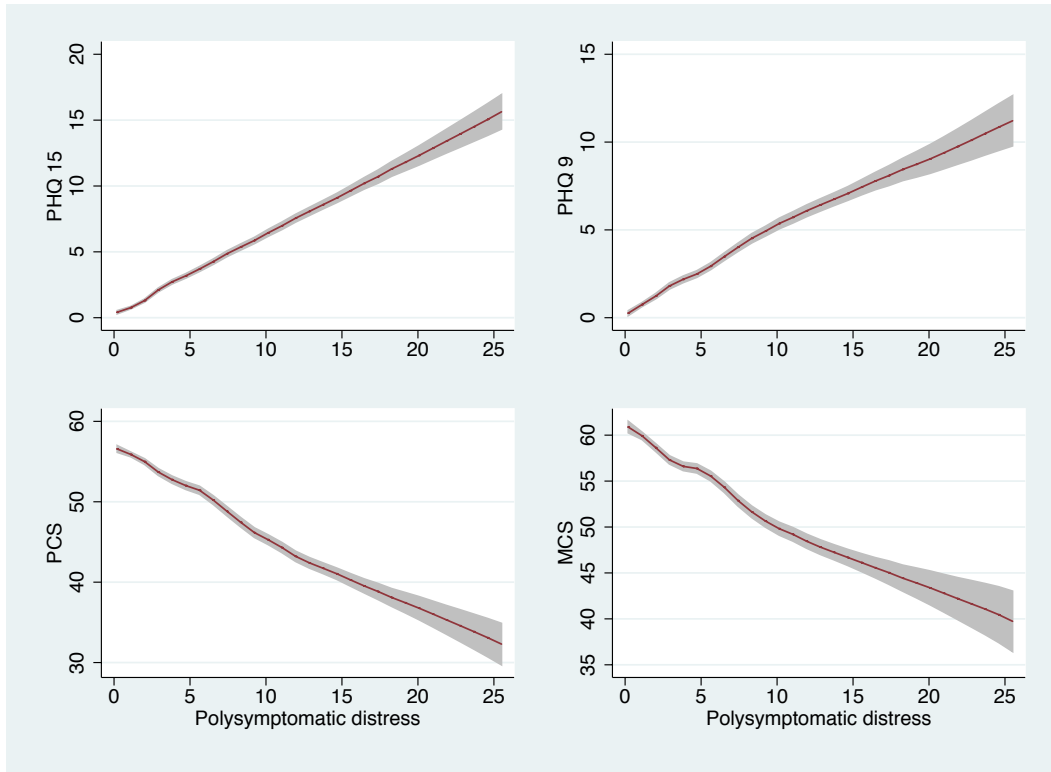


Figure 4. The relation of PSD to the SF-36 PCS and MCS, PHQ-15 somatic severity index and PHQ-9 depression index in the general population. (Figure 4). Covariates are consistently related to differences in polysymptomatic distress across the full relevant range of the dimensional distribution.

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