
The Diagnostic Making of Fibromyalgia Syndrome

According to Robert Bennett, one of the national figures in fibromyalgia syndrome (FMS) research and treatment, fibromyalgia is a descriptive “construct developed by rheumatologists to account for a common group of patients that they see in their routine practice” (Bennett 1999a: 1). Although FMS originated as an intellectual construct developed by rheumatologists to help in the study and treatment of a common group of patients, the idea of FMS has since become reified at the level of both knowledge and experience. Much of this book is devoted to describing the phenomenon of FMS at the experiential level. But before those who suffer could come to organize their experiences around the diagnosis of FMS, the diagnosis itself had to be made and formalized as biomedical knowledge. This chapter describes how rheumatologists developed the construct or idea of FMS and the subsequent debate about that idea within the institutional field of biomedicine, generally, and the subspecialty of rheumatology, in particular.

The Subspecialty of Rheumatology

Rheumatology has been, and continues to be, the field of medicine most closely associated with FMS. Rheumatology is a subfield of internal medicine and, in general terms, its claim of professional expertise involves conditions of the musculoskeletal system. Although current biomedical science rejects the premise that FMS is a musculoskeletal disorder, those who

suffer from it experience intense muscle and joint pain, which places rheumatologists at the center of the FMS story.

Rheumatology as a field of American medicine emerged between the 1920s and 1940s (Kersley and Glyn 1991; Smyth et al. 1985). Following the lead of efforts underway in Europe, Dr. Ralph Pemberton was the first American physician to call himself a rheumatologist and limit his practice to rheumatic disease. In 1926, Pemberton opened the first arthritis clinic in the United States at Pennsylvania's Presbyterian Hospital in Philadelphia. At roughly the same time, several prestigious medical schools, including Harvard and Columbia, initiated academic departments of rheumatology, bringing attention and respectability to the enterprise through their residency training. Paralleling these practical and educational ventures were efforts to create a professional organization. In 1927, the American Committee for the Control of Rheumatism was formed, which ten years later changed its name to the American Rheumatism Association (and would later still change its name to the American College of Rheumatology). During the 1940s, as its membership grew, the American Rheumatism Association (ARA) began to have a discernible professional impact. For example, a dramatic indication of professional consolidation occurred in 1941, when the ARA developed and adopted the first classification for rheumatic diseases. In 1947, the American Rheumatism Foundation (now called the Arthritis Foundation) was created in partnership with the ARA to provide research funds to clinician researchers, enticing a new generation into the field. By the end of the 1940s, a nascent field of rheumatology was well underway.

In contrast to the steady progress in the organizational development of rheumatology between the mid 1920s and 1950, there was little corresponding success in the treatment of most rheumatic diseases. Between 1880 and 1950, the curative promise of medicine was found in the field of bacteriology. As such, rheumatic disorders were primarily treated as though infectious in origin. This approach did not result in many successes, although a few exceptions existed, including the elimination of arthritic conditions caused by gonorrhea, syphilis, and tuberculosis. Additionally, observations linking hemolytic streptococci and rheumatic fever among soldiers in WWII led to the near total elimination of this disease in the United States (Smyth et al. 1985). Overall, however, the developing field of rheumatology had little to boast of in terms of its general therapeutic efficacy until the end of the 1940s.

In 1949, researchers discovered the therapeutic benefits of cortisone for the treatment of arthritis. Even taking into consideration the negative side effects of cortisone and its synthetic siblings, this development represents the zenith of rheumatology's therapeutic promise. The introduction

of corticosteroids to treat rheumatic inflammation is such a significant benchmark in the development of rheumatology that many divide the field's history in terms of "BC" and "AC" (*before cortisone* and *after cortisone*) (Kersley and Glyn 1991: 84). The therapeutic euphoria in the immediate AC period facilitated the establishment of the Institute of Arthritis and Metabolic Disease within the National Institute of Health in 1950. Along with Arthritis Foundation monies, research funds from the Institute added considerably to rheumatology's professional potential and allure.

During the next several decades, there were other significant signs of rheumatology's institutionalization as a specialty in American medicine. In 1958, for example, the ARA established its official journal *Arthritis and Rheumatism*, thereby creating a specialty-based forum for peer-reviewed scientific exchange. This journal remains the most cited rheumatology journal worldwide (Weinblatt 2002). Another watershed for rheumatology was the establishment of board certification. The first examination for certification in rheumatology was given in 1972 by the American Board of Internal Medicine, thus formalizing rheumatology as a subspecialty. In many regards, this step represented the completion of rheumatology's formal professional development. In the language of the sociology of professions, it had formally established its professional jurisdiction (Abbott 1988; Freidson 1971; Larson 1977). Rheumatology had a unique and legitimate claim of professional expertise and it created a gate-keeping mechanism to ensure that only trained and certified clinicians had legitimate access to that jurisdiction.

Rheumatology is now a small, but recognized, field in contemporary American medicine. There are approximately 4,000 board-certified rheumatologists, many of whom are members of rheumatology's professional association, the American College of Rheumatology (ACR) (American Board of Internal Medicine 2004).¹ Yet, in many regards, the field of rheumatology remains professionally precarious. One important reason for its precariousness is that it has few clear answers or solutions for the wide range of illnesses that fall under its jurisdiction. The common conditions that bring patients to rheumatologists—various types of arthritis, lupus, osteoporosis, and fibromyalgia—are dissimilar, chronic, and complex. In fact, many of the disorders that fall to rheumatology are not musculoskeletal disorders at all, despite the fact that the rheumatology's professional expertise involves the musculoskeletal system. Accordingly, rheumatology has no overarching conceptual paradigms or unified principles to organize intellectually the illnesses and treatments of its routine sphere of authority. It is not inaccurate to say that most rheumatological diseases continue to be poorly understood and poorly managed.²

For example, rheumatology's contemporary therapeutic repertoire includes a collection of unsatisfactory options. In addition to a host of newer steroidal drugs, countless nonsteroidal anti-inflammatory drugs (NSAID) are now available. The sheer number of steroidal and NSAID options indicates that not one is fully satisfactory, either in terms of effectiveness or safety, for the treatment of rheumatic patients (Weinblatt 2002). Although current excitement exists about new disease-modifying anti-rheumatic drugs (DMARD), which through immune suppression may slow the progression of rheumatoid arthritis, they do not cure the underlying pathology and they bring a host of problematic side effects. In sum, clinical practice in rheumatology primarily involves partially managing patient's symptoms, whereas the goal of eradicating rheumatologic illnesses remains as elusive to the contemporary rheumatologists as it did to Ralph Pemberton, the father of American rheumatology, in the 1920s.

Unquestionably, the factors outlined above contribute to the current gloomy mood of the subspecialty. A palpable sense is found among its practitioners that rheumatology is facing hard times. There are still very few rheumatologists. Moreover, the number of trainees entering rheumatology programs is falling, suggesting that their numbers will only decline further. That rheumatology programs are not widely represented at academic medical centers both illustrates and perpetuates the field's failure to thrive. Additionally, the managed care environment presents ever greater threats. The cost-benefit model that characterizes the new era of health outcomes research bodes poorly for a subfield dependent on justifiable referrals when patient improvement, as a rule, is negligible in relation to the expense. Likewise, health economists have determined that rheumatology clinics do not benefit a hospital's bottom line, making its future in corporate run (and nonprofit, but corporate-like run) health care uncertain (Weinblatt 2002).

As a brief aside: Some of the struggles facing rheumatology parallel those facing other specialists and, to some degree, the medical profession as a whole. For example, today all physicians face complex, chronic illnesses that are often poorly understood or poorly managed. Likewise, rheumatology is not the only specialty whose overarching conceptual focus fails to account meaningfully for the range of conditions that fall under its purview. By contrast, the most prestigious specialty—surgery—has a very clearly defined conceptual jurisdiction.³ Lastly, all specialties and, indeed, all medical practitioners, have felt the impact of dramatic changes in the health-care delivery system during the last several decades brought on by managed care. None of these parallels, however, belie

the foregoing characterization of rheumatology as a relatively marginal professional player within the institution of medicine and a subfield marked by few glamorous theoretical or therapeutic payoffs.

In this highly condensed historical account of rheumatology, a key detail has been intentionally omitted. Whereas the subfield of rheumatology was established to study and treat disorders of the musculoskeletal system, it has come to treat patients primarily on the basis of the symptomatic experience of pain. Much has been said and written about pain's biomedical elusiveness (Scarry 1985). No objective evidence of pain exists; only a patient's subjective testimony. In this way, pain is "a medical object distinct from those that can be directly read from the body or discovered through laboratory tests" (Baszanger 1995: 8). As a result, the world of biomedicine has not treated pain as an end in and of itself (much to the regret of chronic pain sufferers), but rather as a diagnostic clue or tool (Baszanger 1995; Morris 1991). Clinicians trace pain backward, so to speak, to the true object of biomedicine: the organic condition producing the experience of pain. Such a strategic stance vis-à-vis pain can be understood, given its elusive and nonparadigmatic qualities. Biomedicine seeks a certainty that pain simply fails to surrender.

Although pain stands at the margins of biomedicine, it stands at the center of rheumatology. In effect, pain has become the conceptual justification for rheumatology. More than anything else, this fact accounts for rheumatology's imprecisely defined professional jurisdiction, relatively inglorious history marked by only modest therapeutic successes, and lack of prestige relative to many other medical specialties. The professional limitations of rheumatology, therefore, are largely a result of the profession's close association with pain, its explanation, and its treatment. For example, because pain falls within rheumatology's jurisdiction, the subfield has found itself historically and contemporarily facing a mass of patient referrals that share, with any certainty, only one thing—the subjective experience of bodily pain. In some cases, rheumatologists have been able to trace pain backward to reveal its organic foundations, but in many cases this strategic approach bears no fruit. Consequently, much of the clinical content of rheumatology is this unsorted, residual patient mass (Kersley and Glyn 1991: 79), and the attempt to create a paradigm from such a heterogeneous patient mass represents the ongoing professional challenge of rheumatology.

From the vast and disparate residual category of patients sent to rheumatologists, the diagnostic category FMS emerged and, as such, FMS illustrates rheumatology's historical relationship to, and struggle with, pain more generally. The following remarks, which appear in a special

issue of *Balliere's Clinical Rheumatology* devoted to the fibromyalgia debate, illustrate the spirit of this ongoing enterprise.

[R]heumatologists, perhaps more than other clinical specialists, have always seen pain in itself, and the associated disability, as a proper part of their remit, beyond the demands of diagnosis. The *Annals of Rheumatism* in the 1940s, for example, contain many wide-ranging descriptions of people in pain whose detailed histories related to war traumas and the psychosocial circumstances of their lives. Whether the designation of “psychogenic rheumatism” in many of these cases was helpful or not is less important than the powerful sense communicated by these case-studies, of concern for the individual’s distress beyond the need for a rheumatological diagnosis (Croft and Silman 1999: xi).

But a need and demand arose for the rheumatological diagnosis of fibromyalgia and its creation must be seen as part of a central professional struggle of rheumatology: giving medical representation to individuals’ subjective distress. Leading FMS expert, Don Goldenberg, explains: “Fibromyalgia is simply a label to use when patients have chronic, unexplained diffuse pain” (Goldenberg 1999: 781). Goldenberg speaks from authority, because he was among a small handful of rheumatologists who originally developed the label “fibromyalgia” and formalized it as a category of biomedical knowledge that now gives representation to the subjective suffering of millions.

The Character of Biomedical Diagnoses

Diagnosing a patient’s distressing symptoms is at the heart of biomedical practice. Crucial for both physician and patient, a diagnosis represents the physician’s comprehension of the patient’s experience. Importantly, a diagnosis gives the physician a medical plan of action to treat or aid the patient in his or her return to health. For the patient, a diagnosis gives meaning and legitimacy to worrying symptoms and provides a framework for what he or she is facing. For all these reasons, when a patient presents distressing symptoms to a physician, both parties may find even a bad diagnosis better than no diagnosis at all (Rosenberg 1997: xviii). A diagnosis effectively legitimizes both parties and the doctor-patient relationship itself.

Medical nomenclature is the collection of diagnoses available to physicians. More than just an unorganized list of disease and disorders, nomenclature is a framework of classification that organizes and represents biomedicine’s formal knowledge (Abbott 1988). For example, diseases are classified together in terms of bodily organs or systems, shared

etiology, pathophysiology, and the like. Such a system of classification represents, among other things, a map of the medical profession's jurisdiction: it represents the conditions over which biomedicine has a legitimate claim of authority and expertise. Nomenclature is not a fixed system but rather a continually revised expression of biomedical knowledge. The consistent trend in nomenclature is toward an increasing number of diagnoses and classification categories (as well as subdiagnoses and subclassifications)—a trend highly apparent in the historical expansion of rheumatological nomenclature.⁴

Historian Charles Rosenberg remarks that a disease does not exist “until it is named” (Rosenberg 1997: xiii).⁵ In a similar vein, sociologist Phil Brown (1995) proposes that a disease comes into existence only with the creation of a diagnostic category. Of course, neither Rosenberg nor Brown denies the material existence of disease outside of its diagnostic naming. Instead, their shared claim is that a disease does not exist *socially* until the social institution of biomedicine names it. In practice, the creation of a new diagnostic name simultaneously declares the existence of a condition and legitimizes biomedicine's professional claim of authority and expertise in relationship to that condition (Freidson 1971). The creation of a new diagnosis, and the inclusion of that diagnostic name within medical nomenclature, represent an *expansion* of biomedicine's professional jurisdiction and might, therefore, be labeled *entrepreneurial*.

The professional task of biomedicine is to determine whether the unorganized and subjective symptoms of patients can be organized and measured in terms that fall within its jurisdiction. It is through the practices and principles of diagnostic research that medicine makes (or fails to make) a new diagnostic category and, therefore, expands (or fails to expand) its jurisdiction. In particular, case-controlled clinical studies comparing individuals believed to have a condition (patients) with those lacking the condition (controls) is a dominant practice used in diagnostic research. This is a simple and familiar research design. A disorder's existence depends on whether it can be translated into biomedical *markers* present among the patients but absent among the controls. The marker or combination of markers with the best balance of sensitivity (the ability to select those with the disorder or “true positives”) and specificity (the ability not to select those without the disorder or “true negatives”) are considered to have the best accuracy in discriminating between patients and controls and, therefore, are favored as diagnostic criteria. In the ideal case, a specific organic marker, a *sign*, confirms a specific diagnosis and is referred to as a diagnostic “gold standard.”

Of course, not all diseases or conditions have diagnostic gold standards. One can think of a continuum of diagnostic certainty ranging

from diagnoses based on clear and present biophysical signs (either directly or indirectly visible through technoscientific methods) to those that rely heavily on descriptive and interpretive evidence. Although much everyday medical practice involves fairly unambiguous diagnostic work, of greatest sociological interest are those that fall on the latter end of this continuum. Because these descriptive diagnoses are often medically and socially contested, they raise interesting questions about biomedicine's contemporary cultural authority and power (Brown 1995).⁶ In short, in the absence of clear biophysical evidence for disease, how and why are diagnoses constituted? The case of FMS is illustrative.

The Emerging Idea of Fibromyalgia Syndrome

The medical literature frequently suggests that fibromyalgia has existed throughout time. Biblical passages, as well as ancient medical references, are used to reveal the historical continuity of the disorder (Smythe 1989; Wallace and Wallace 1999). Despite this presumed ancient lineage, it is widely recognized that a few key publications concerning the rheumatic condition "fibrositis" in the 1970s marked a critical turning point in the contemporary framing of FMS.

Stage One: The (Re)introduction of Fibrositis

Fibrositis is a condition with a long history of medical marginality.⁷ Sir William Gowers first introduced the term in 1904 in an article on lumbago in the *British Medical Journal*. The term "fibrositis," which means inflammation of fibrous tissue, was quickly judged erroneous; fibrous tissue cannot become inflamed. Nevertheless, the term persisted in medical parlance and was used to describe unexplainable, and oftentimes exaggerated, muscle and joint pain, including, for example, its application to shell-shocked soldiers in WWII. An article published in 1947 in the *Annals of Rheumatic Disease* argued that fibrositis be considered a form of "psychogenic rheumatism," given that those who suffered displayed no inflammation but had high rates of depression and anxiety (Boland 1947). This sentiment was widely adopted. During most of the twentieth century, fibrositis was essentially synonymous with psychogenic rheumatism, a belief that persisted and became entrenched in the minds of most physicians.

Such was the state of affairs in 1972 when rheumatologist Hugh Smythe wrote a chapter on fibrositis for the widely used textbook, *Arthritis and Allied Conditions*.⁸ Smythe (1972) described fibrositis as a long-recognized and poorly understood rheumatic condition, but insisted that a distinct form of the disorder could easily be distinguished from that with

psychogenic origins. In particular, unlike the *bizarre* forms of pain and paralysis seen with psychogenic rheumatism, Smythe contended that patients with fibrositis had truly *mundane* pain and that they could be distinguished by the presence of tenderness in characteristic locations. Smythe called these locations “tender points” and proposed that they be used as the key element in a set of diagnostic criteria to identify patients with fibrositis.⁹

The idea that tender places on the body are associated with muscular rheumatism has been propagated in various forms since the early 1900s. These tender places were called “nodules,” “nerve points,” “trigger points,” and “tender points” by different researchers at different times during the twentieth century. As the existence of many different terms implies, a clear sense about the nature of these tender places and their biophysical existence has never been substantiated (Reynolds 1983). Nevertheless, Smythe employed the medically speculative concept of tender points to carve out a clinically identifiable pain syndrome from a collection of vague and diffuse symptoms described in the medical literature for at least two centuries.

By conceptually extracting a new and distinct medical entity from the residual category “psychogenic rheumatism,” Smythe played a central entrepreneurial role in the diagnostic making of FMS. The response to Smythe’s entrepreneurial effort was the burgeoning of fibrositis research as other clinician researchers eagerly set out to understand and treat this newly identified medical entity. Although Smythe’s detailed description of fibrositis and his proposed criteria were significant, he had yet to test the criteria clinically. Was it possible to identify patients with fibrositis as Smythe suggested? Was there a subset of patients who could be conceptually disentangled from the amorphous category of psychogenic rheumatism? Answering these questions was the next stage in the diagnostic making of FMS.

Stage Two: The Entrepreneurial Challenge

During the 1980s, a small number of diagnostic entrepreneurs picked up where Smythe left off. The key players were four university-based rheumatologists: Muhammad Yunus (Peoria School of Medicine), Robert Bennett (Oregon Health Sciences University), Frederick Wolfe (University of Kansas School of Medicine), and Don Goldenberg (Boston University School of Medicine). Each had his own patients with ill-specified symptoms and multiple tender points as did Smythe’s patients with fibrositis. This new cohort of diagnostic entrepreneurs favored the term “fibromyalgia”. After all, their patients had no muscle and tissue inflammation and fibromyalgia better captured what they considered the disorder’s

cardinal symptom: muscle and joint pain. With a new name in hand, they set out to determine if it was possible to distinguish their patients with fibromyalgia from controls in clinical studies. Was fibromyalgia a clinical entity they could measure and study and, therefore, hope to treat?

Rheumatologists Yunus, Bennett, Wolfe, and Goldenberg, each with his respective research team, clinically tested and published fibromyalgia criteria. They used case-controlled clinical studies to find the precise combination of symptoms that best adjudicated between patients they identified as having fibromyalgia and controls. As the 1980s came to a close, however, the results of their collective research efforts were troubling and paradoxical. Each of the research teams published clinically tested FMS criteria, but their criteria differed. They all proposed tender points as criteria but disagreed about their location and how many were required for diagnosis. They also disagreed on which, if any, of the symptoms commonly associated with fibromyalgia (e.g., fatigue, sleep disorders, irritable bowel syndrome) were diagnostically necessary. The lack of consensus in establishing criteria fed into the well-entrenched sense that, whether the condition was called “fibrositis” or “fibromyalgia syndrome,” it was an ill-specified diagnosis at best.

Stage Three: The American College of Rheumatology Criteria

To address the heterogeneity of existing criteria sets, the diagnostic entrepreneurs agreed to come together, collectively design and conduct a study, and identify the single best set of diagnostic criteria for fibromyalgia. Rheumatologists from a total of sixteen research centers joined their effort. This group, which became the American College of Rheumatology (ACR) Multicenter Fibromyalgia Criteria Committee, eventually proposed the fibromyalgia diagnostic criteria that were formally approved by the ACR.

In brief, the Committee research proceeded as follows. First, each of the participating research centers contributed a number of patients it identified as having fibromyalgia and a designated number of controls. To correct for earlier variations in data collection, researchers gathered standardized data on patients and controls at each participating center and then determined what factors best distinguished patients from controls. Based on a series of statistical combinations and comparisons, the Committee concluded that multiple tender points were the “most powerful discriminator between fibromyalgia patients and controls” (Wolfe et al. 1990: 166). In addition, they found that widespread pain (defined as pain in all four quadrants of the body) was found in 98 percent of patients with fibromyalgia, but in only 69 percent of controls. Whereas tender points had high levels of specificity, widespread pain was highly

sensitive. The Committee combined these two features in its proposed criteria: eleven or more positive tender points (of eighteen test points), in combination with widespread pain, offered the most sensitive, specific, and accurate criteria for the diagnosis of FMS. Using these criteria, 88 percent of patients with fibromyalgia were identified as having fibromyalgia (true positives), and 81 percent of controls were identified as not having fibromyalgia (true negatives).

The ACR study, thus, established a category of patients who could be recognized in a clinical context with a high level of accuracy. Fibromyalgia, the Committee concluded, is an identifiable clinical entity, and the uniform diagnostic criteria it established were advanced and adopted by the ACR in 1990. The published account of the Committee's research appeared in *Arthritis and Rheumatism*, the official journal of the ACR.

Stage Four: FMS Post-ACR

Between 1972 and 1990 much and little had changed with respect to fibrositis syndrome. Smythe's initial specification of criteria in 1972 brought new attention to a common and poorly understood set of symptoms that had long brought a steady stream of patients to rheumatology and general outpatient clinics. His criteria were tested, modified, tested again, and eventually evolved into the fibromyalgia diagnostic criteria formally adopted by the ACR. But identifying criteria, formally adopting the diagnosis into medical nomenclature, and changing the disorder's name did not result in medical consensus. Despite the ACR Committee's best intentions to move beyond the contested history of psychogenic rheumatism and fibrositis, what remained the same, by any name, was the disorder's lack of an objective biomedical sign or marker.

The diagnosis of FMS is dependent on the subjective report of patients. Tender points do not represent fibrous tissue or muscle pathology and, therefore, do not represent anatomical pathology (Block 1999). The ACR Committee's elaborate analysis did not establish objective criteria. Instead it formalized a standard set of subjective criteria derived through clinical observation. Moreover, the ACR's analysis was built on a methodological flaw, set in motion by Smythe, and reproduced in every subsequent FMS diagnostic study. FMS is a *tautology*: tender points both define and substantiate its existence. Stated simply, the diagnostic entrepreneurs compared patients with FMS (defined by the presence of a large number of positive tender points) with controls (individuals without a large number of positive tender points) and found, over and over again in study after study, that a large number of positive tender points best distinguished patients with FMS from controls.¹⁰ According to one of the most outspoken FMS

critics, although the ACR study generated no shortage of statistics, nothing could save it from having already “succumbed to a ‘garbage in/garbage out’ problem” (Bohr 1996: 594).

If FMS was not controversial enough, in 1992 a working group at the Second World Congress on Myofascial Pain and Fibromyalgia, which included many of the diagnostic entrepreneurs, proposed flexibility in the diagnostic criteria in a document titled the *Copenhagen Declaration*. An article in the British medical journal *Lancet* (Csillag 1992: 663–64) quotes from the *Copenhagen Declaration*:

[T]he diagnosis is commonly entertained in the presence of unexplained widespread pain or aching, persistent fatigue, generalized [morning] stiffness, non-refreshing sleep, and multiple tender points. Most patients with these symptoms have 11 or more tender points. But a variable proportion of other typical patients may have less than 11 tender points at the time of the examination.

Thus, the group recommended clinical flexibility in the tender point requirement for patients who otherwise exhibit some of the symptoms that cluster as part of a wider syndrome: “encompassing headaches, irritable bladder, dysmenorrhoea, cold sensitivity, Raynaud’s phenomenon, restless legs, atypical patterns of numbness and tingling, exercise intolerance, and complaints of weakness” (Csillag 1992: 664). The recommendations outlined in the *Copenhagen Declaration* were subsequently incorporated into the World Health Organization’s tenth edition of *International Statistical Classification of Disease and Related Health Problems* (1993). As a result, strictly speaking, there are no necessary or sufficient criteria (the tender point count can be flexible) or required number of associated symptoms (no particular number was specified in the *Copenhagen Declaration*) for FMS to exist. In practical terms, this opens the door for tremendous variation in how individual practitioners diagnose conditions in individual patients in clinical settings, leading some critics to argue that it is nearly impossible not to arrive at a diagnosis of FMS for every patient with widespread pain and tenderness of unknown origin (Cohen 1999).

These criticisms notwithstanding, FMS has been endowed with social life and that life continues to unfold. Favorably inclined rheumatologists and other medical practitioners have a new diagnosis for their dealings with a large and persistent patient population and millions of sufferers have now had their conditions diagnosed. The diagnostic making of FMS has also created a new professional niche of treatment and research. Diagnostic entrepreneur Robert Bennett summarizes the story thus far

and, in so doing, outlines both the professional problems and promise FMS represents.

Fibromyalgia is a clinical construct that has been developed, for the most part, by rheumatologists. It is a direct descendent of “fibrositis,” a common misnomer that was first coined in 1904. There are always problems inherent in defining a disorder in purely descriptive terms. Nevertheless the publication of the American College of Rheumatology’s 1990 Classification Criteria for fibromyalgia has been coincident with an impressive resurgence of research in this area (Bennett 2003: 5).

FMS: A Body of Knowledge¹¹

Even before the ACR criteria were formally established, the efforts of the diagnostic entrepreneurs drew enough attention to promote interest among other clinician researchers. Slowly at first, but then with increasing pace, additional research appeared in the medical literature. By the end of 2000 more than 1,000 fibromyalgia publications appeared in the medical literature, most of them in one of thirteen rheumatology journals.¹² Figure 1.1 illustrates the historical trajectory of these publications, breaking them down by type: research articles ($n = 627$), review articles ($n = 179$), and comments or editorial pieces ($n = 220$).

As already outlined, the few publications in the early years focused primarily on describing the clinical entity of fibrositis and reflecting on its long history under various, now medically anachronistic, names such as psychogenic rheumatism and lumbago. The number of research publications picked up appreciably, beginning in the mid 1980s, as earlier descriptive work gave way to the second wave of diagnostic entrepreneurship, including the clinical testing of proposed diagnostic parameters. By the mid 1980s, these efforts prompted others to investigate the disorder’s pathophysiology, etiology, and treatment. Correspondingly, from the mid 1980s onward, a substantial number of review articles appeared, indicating the emergence of an area or body of research considered worthy of systematic appraisal and critique. These trends become even more pronounced in the wake of the adoption of the ACR criteria in 1990. Finally, the steady rise in comment and editorial publications reveals the debate—assertions and counter-assertions concerning the scientific basis of the diagnosis and disorder—that has become increasingly intense.

One can summarize the divide within the body of FMS knowledge into two overarching positions. First, those who argue that FMS is an organic disorder or a disorder with a central organic component, and second,

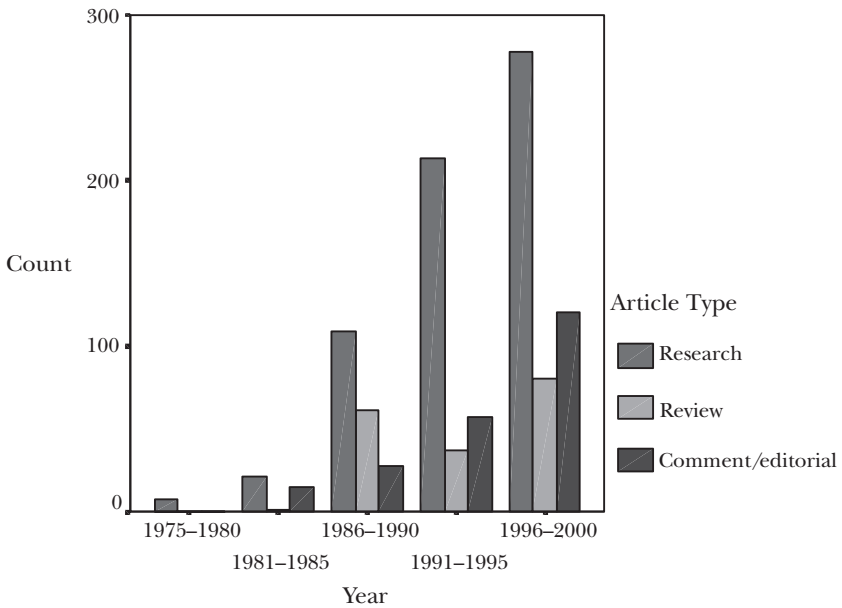


Figure 1.1 Fibromyalgia Publications in the Medical Literature, 1975–2000.

those who claim the disorder is a psychogenic or behavioral disorder, some of whom reject the scientific merit of the diagnosis itself. In practice, these camps are far from internally monolithic because extreme and moderate positions are found within each. Moreover, these camps are not truly mutually exclusive or exhaustive. Some researchers interpret FMS as a neurobiological psychiatric disorder, but suggest that current psychiatric classifications be used in lieu of the FMS diagnosis. Also blurring the division, very few in the organic camp would argue that psychological and behavioral factors play no role in FMS, and some attempt to bridge the organic/psychogenic divide by promoting a “biopsychosocial” account of FMS. Despite the complexity of positions, the crux of the debate over FMS is essentially a division between those who believe FMS is linked to some aberrant physiology and those who do not.

This divide can be seen in the FMS research literature. By far the most common types of FMS publications (almost half of all research articles) are those that explore factors thought to be associated, possibly causally, with FMS (see Appendix A for more details). Figure 1.2 illustrates the five most common factors studied in FMS association or causation research in terms of numbers of publications over time. Four of these are organic pursuits, whereas the fifth encompasses research testing a host of psychological

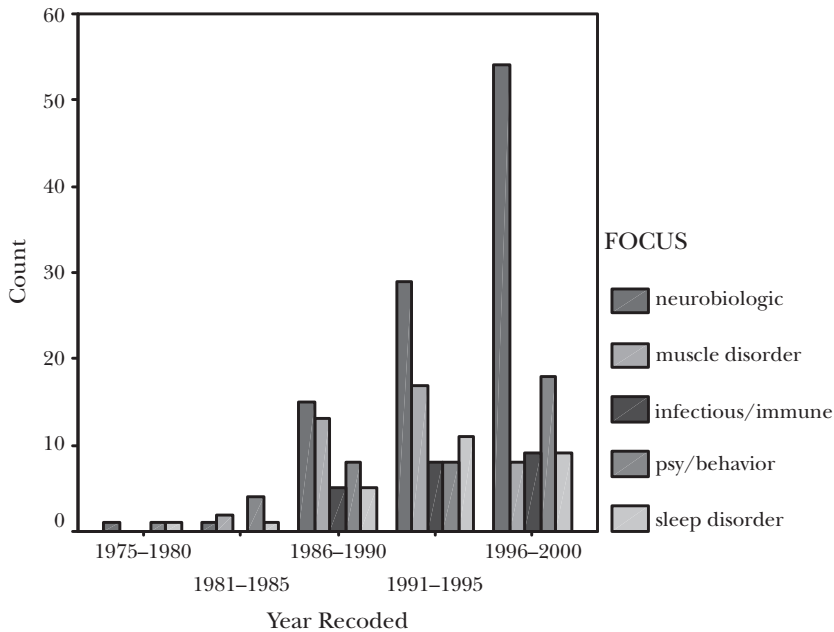


Figure 1.2 Focus of Fibromyalgia Publications, 1975–2000.

or behavioral hypotheses. The general knowledge claims of these camps will now briefly be summarized.

Organic Pursuits

Given the musculoskeletal nature of FMS pain, it is not surprising that significant effort has been directed toward finding abnormalities in muscle tissue of those who sufferer such pain. Studies in the 1980s looked promising. Some found evidence for structural irregularities in muscle and related soft tissue in patients with FMS, whereas others found the muscles of those with FMS to have diminished energy metabolism. These studies were poorly designed, however, and all subsequent attempts to find muscle abnormalities failed (Block 1999). The collective results from more than two decades of research demonstrate no muscle or soft tissue pathology, either in structure or performance, with FMS, although this has hardly curbed the efforts of some clinician researchers to prove otherwise (Block 1999).

Similarly persistent have been attempts to establish a link between sleep irregularities and FMS. In 1977, concurrent with his attempts to specify fibrositis criteria, Smythe and a colleague at the University of

Toronto published an account of fibrositis as “a non-restorative sleep syndrome” characterized by electroencephalograph (EEG) images of disturbed stage-4 or non-rapid eye movement (REM) sleep (Smythe and Moldofsky 1977). Since the 1970s, several studies have demonstrated differences in the brain wave activity in non-REM sleep of patients with FMS compared with healthy controls. One recent study found these abnormalities predictive of FMS-symptom severity (Roizenblatt et al. 2001). Although promising, these findings lack sensitivity and specificity; many individuals whose condition is diagnosed as FMS have no such abnormalities, and such abnormalities are both associated with other disorders and widely present in the general (healthy) public. After twenty-five years of ongoing research in this vein, no convincing evidence yet links FMS to any specific and objectively measurable sleep abnormalities (Cohen 1999). As with muscle pathology research, however, the lack of promising findings has not deterred researchers who continue to pursue links between FMS and sleep irregularities—perhaps spurred on by the fact that nearly all those whose condition is diagnosed as FMS report nonrestful sleep and morning fatigue (Abeles 1998).

Research exploring the link between FMS and infectious illness has also met with little success. More than two dozen review and research articles evaluating a possible association between infectious illness and FMS have been published, but no convincing link has been established (Goldenberg 1999). It is generally accepted that FMS does not have an immune component, although many sufferers associate the onset of their symptoms with a prolonged infectious illness and report an ongoing subjective sense of immune deficiency.¹³ Once again, persistent, nonsupportive findings have not brought an end to this line of inquiry.

Figure 1.2 dramatically reveals the current emphasis of FMS research. An increasing number of clinician researchers argue that fibromyalgia is a neurobiological disorder. In contrast to the above-noted dead ends, this is currently the most promising organic account of the disorder. Several of the diagnostic entrepreneurs, including Bennett and Goldenberg, describe FMS as a disorder characterized by aberrant central pain processing. In particular, it is argued that individuals with FMS experience nonpainful stimuli as painful (called allodynia) and have an exaggerated response to painful stimuli (called hyperalgesia). It is assumed, therefore, that FMS involves an abnormality in nociception, the process by which painful stimuli are transmitted neurochemically between the peripheral and central nervous systems.

Several possible explanations have been advanced for why the body's system of pain perception malfunctions but, in general, they all involve various notions of neurochemical cross circuiting. Somewhere within the body's system for pain perception, which includes the peripheral nerves,

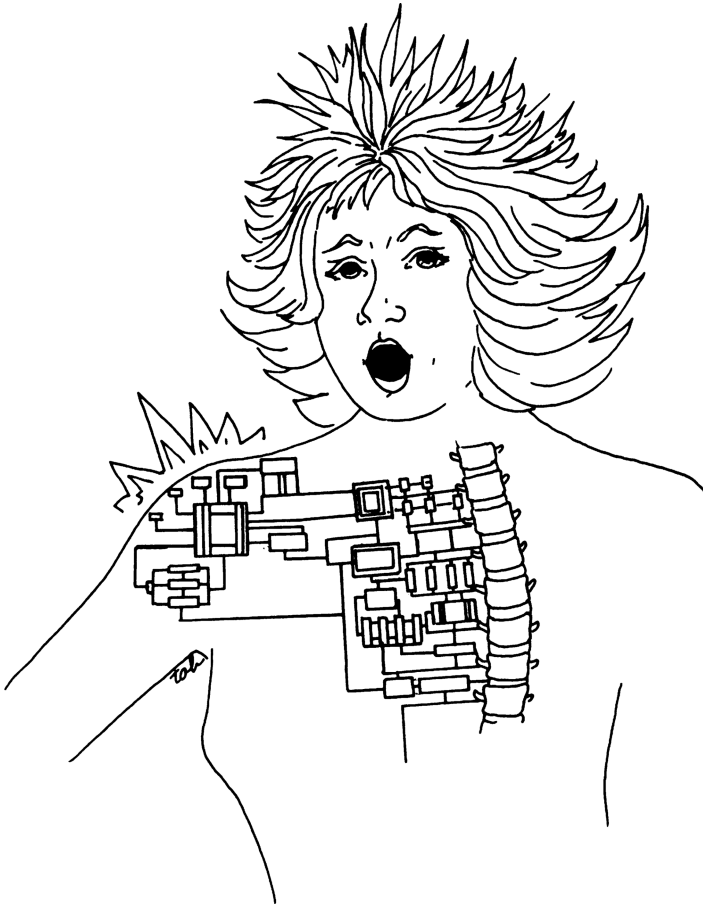


Figure 1.3 Graphic Depiction of Short-Circuiting (Wallace and Wallace 1999: xiv).

the spinal cord, the brain, and specific neurotransmitters, neurochemical misinformation is sent or processed. The body becomes hyperresponsive to pain, even in the absence of painful stimuli. Figure 1.3 from an FMS self-help book sponsored by the Arthritis Foundation, provides a simple visual representation of nociceptive malfunctioning (or a short-circuiting) in the central nervous system that results in an alteration in pain processing.

A leading explanation for why patients with FMS have such short-circuiting involves a phenomenon called “wind-up” (Bennett 1999b). In simple terms, wind-up is the neurochemical outcome of chronic pain. When pain lasts long enough and is intense enough, the argument goes, it tricks the body’s neurochemistry into perceiving pain when there are

no painful stimuli. Ordinarily, the experience of acute pain is in proportion to painful stimuli; when we get injured, we experience pain; as the injury heals, the pain too subsides. Researchers argue that this framework does not apply to *chronic* pain states wherein no direct link is found between an injury (painful stimuli) and the experience of pain. Therefore, rheumatologists are unable to trace pain back to any physical injury or anatomic pathology. Advocates of this stance claim that once the neuroscience of chronic pain states are more widely acknowledged by medical orthodoxy, fibromyalgia, which is said to occupy the far end of a spectrum of chronic pain, will no longer be controversial.

This model of FMS as a disorder of pain amplification is intuitively and culturally compelling. In addition, it accounts for the defining criterial feature of FMS: the characteristic presence of multiple tender points. A large number of tender points that respond to low levels of stimuli suggest an alteration in pain perception. But more persuasive yet, this model theoretically integrates empirical evidence purportedly demonstrating an association between FMS and neurobiological abnormalities.

For example, it is claimed that FMS is related to irregularities in two neurotransmitters involved in nociception—serotonin and substance P. Metaphorically, neurotransmitters are understood as the brain's chemical messengers. As a neurotransmitter involved in the body's pain response, substance P sends pain signals to the spinal cord that, in turn, communicates the message of pain to the brain. Accordingly, too much substance P in the body could result in the perception of nonpainful stimuli as painful (allodynia) and an exaggerated response to painful stimuli (hyperalgesia). Several studies that have found elevated substance P levels in the spinal fluid of those with FMS are routinely marshaled in support of the neurobiological model. Although interesting, an elevation of substance P is found in many other conditions in which pain is a central feature and, therefore, is not specific to FMS (Cohen 1999).

Whereas substance P encourages the communication of pain messages to the brain, it is argued that the neurotransmitter serotonin helps regulate or discourage the message of pain. Therefore, too little serotonin (or diminished serotonin effect) could lead to fairly harmless stimuli being experienced as painful (hyperalgesia). In line with this argument, studies that have found an association between FMS and compromised serotonin levels and metabolism are heralded as additional proof of neurobiological causation (Russell et al. 1992). As will be explained shortly, however, others interpret these same findings as evidence that FMS is merely the somatic presentation of depression. Making the matter more complex yet, recent research has failed to replicate the association between serotonin uptake and fibromyalgia (Legangneux et al. 2001).

Other empirical evidence said to confirm the neurobiological basis of aberrant pain processing in FMS comes from reports of brain abnormalities detected through imaging technology. Researchers claim that FMS is characterized by single-photon emission computerized tomography (SPECT) images that show decreased blood flow to a location in the brain involved in nociception (Mountz et al. 1995). This research is criticized as being riddled with methodological flaws, most centrally involving the selection of appropriate controls (Abeles 1998). Research has yet to compare patients with FMS with other chronic pain patients. It is unclear, therefore, if the observed SPECT alterations are unique to FMS or common to pain states more generally. Moreover, these studies have not adequately controlled for psychiatric illness, even though SPECT alterations are all the rage in neuropsychiatry. Consequently, as with linking FMS to serotonin abnormalities, evidence of SPECT alterations can equally support the claim that FMS is principally a psychiatric condition.

In yet another vein, researchers assert that FMS involves neuroendocrine irregularities, in particular, dysregulation in the hypothalamus-pituitary-adrenal (HPA) axis (Crofford et al. 1996). In lay terms, the HPA axis is involved in the body's stress response. Individuals with FMS, it is argued, do not have a normal response to acute physical or psychological stress but, rather, their bodies behave as though they are in a chronic state of stress. This argument is based on observed alterations of certain neurohormones (growth hormone and cortisol) among those with FMS (Crofford and Clauw 2002; Goldenberg 1999). At question, however, are how these neuroendocrine abnormalities associated with stress are linked to FMS symptoms and what causes HPA dysregulation in the first place. For example, psychiatric distress can impact HPA functioning, leading other researchers to use these same findings in support of the psychogenic account of FMS (Abeles 1998).

Based on the combined neurobiological evidence (i.e., neurotransmitter and neurohormonal anomalies), some clinician researchers suggest that FMS is characterized by an abnormality in sensory processing more generally, called "central sensitivity," of which pain processing is but one aspect. A heightened response to a host of stimuli would account for many of the somatic complaints that make up the FMS symptom constellation. Likewise, it would account for the widely recognized overlap between FMS and other controversial syndromes involving sensory sensitivity, such as chronic fatigue syndrome (CFS), multiple chemical sensitive (MCS), irritable bowel syndrome (IBS), and premenstrual syndrome (PMS), to name but a few. Following this logic, some clinician researchers claim that, along with these other syndromes, fibromyalgia is best described as a *dysregulation spectrum syndrome* or a syndrome in which some common

underlying mechanism manifests itself in hypersensitivity to a wide range of sensory stimuli (Yunus 2001: 130).

Even as many of the leading FMS clinician researchers promote an account of FMS as a dysregulation spectrum syndrome or a disorder of central sensory processing, few of them fail to acknowledge the *complex* nature of fibromyalgia, by which they mean the influence of nonbiological factors. Instead, they accept that an important interplay exists between the psychological, social, and biophysical aspects of FMS. Some have misappropriated the term “biopsychosocial” (Henriksson 2002),¹⁴ whereas others suggest that FMS and related syndromes are “stress-associated” to capture the interplay between the body’s biochemical stress response and the everyday lives of individuals (Crofford et al. 1996).

Although it would be false to say that the neurobiological perspective is monocausal, it is fair to say it frames FMS as *primarily* organic with associated psychological factors. For example, although Bennett himself maintains that one would be misguided to approach FMS as either a solely organic or solely psychological problem, it is clear that his orientation favors the former, as does his research agenda. The essence of this perspective, with its ambition to tie together sensory dysfunction and neurobiological abnormalities, is nicely captured in the following quote from Bennett’s report, aptly titled *The Scientific Basis for Understanding Pain in Fibromyalgia*:

Considering the preponderance of studies pointing to a dysfunction of sensory processing in fibromyalgia, one would expect these patients to have an amplification of bodily sensations resulting in a wide range of somatic symptoms. A diagnosis of somataform disorder [a disorder characterized by multiple symptoms that cannot be traced to a physical cause] will become a non-psychiatric diagnosis once the symptomology is adequately explained by disordered physiology (Bennett 2003: 1).

Psychopathological and Behavioral Pursuits

As a diagnostic subset carved out of the residual category of psychogenic rheumatism by Smythe and other diagnostic entrepreneurs, FMS was born under the burden of doubt. Consequently, when rheumatologists were unable to produce solid evidence of an organic basis for the new disorder, it took no time for a psychogenic framing of the disorder to crystallize and build over time (see Figure 1.2). Early on, some clinician researchers claimed that FMS symptoms were the somatic manifestation of psychological distress and they supported these claims with their anecdotal impressions that many of those whose conditions were diagnosed as FMS were mentally unwell. By the 1980s, clinician researchers began to test empirically the anecdotal sense that there was a link between FMS and