

Viewpoint

Fibromyalgia falls foul of a fallacy

John L Quintner, Milton L Cohen

"Diagnosis is a system of more or less accurate guessing in which the end-point achieved is a name. These names applied to disease come to assume the importance of specific entities, whereas they are for the most part no more than insecure and therefore temporary conceptions".¹

Fibromyalgia as a diagnosis was first conceived in 1981 to label a syndrome characterised by widespread pain and increased sensitivity to pressure at various specific anatomical locations known as "tender points".² During the subsequent 10 years, the spectrum of fibromyalgia expanded to include non-musculoskeletal clinical features, such as fatigue, sleep disturbance, headache, and irritable bowel.³ In an attempt to facilitate epidemiological study, the American College of Rheumatology (ACR) announced classification criteria for fibromyalgia in 1990, to distinguish it from other rheumatic conditions.¹ We have previously argued that these criteria were developed out of circular reasoning, with the result that fibromyalgia has become a proposition so broad that it includes all possibilities.⁴ Despite this objection, fibromyalgia seems to be an entrenched diagnostic label—at least in rheumatological circles—but it is a label so easily abused as to have become meaningless.^{5,6} We aim to expose the major flaws in the proposed entity of fibromyalgia, to trace its conceptual evolution, and to propose an alternative model that draws on current understanding of the neurobiology of chronic pain.

Fibromyalgia has been promoted as "a common and recognisable cause of chronic, diffuse musculoskeletal pain".^{7,8} This statement violates the dictum in logic that an effect—in this case an illness—should not be confused with its own cause. Fibromyalgia cannot be both a state of musculoskeletal pain and the cause of that state, which raises the question of whether the label fibromyalgia is intended to imply the existence of a specific entity (an essentialist interpretation), or whether the name is to be used as only a working hypothesis in which certain clinical features are grouped together to test hypotheses of causation (nominalist interpretation).⁹ The acceptance of fibromyalgia as an essentialist truth rather than a nominalist hypothesis has confounded scientific understanding of the phenomena it purports to describe.

The ACR criteria for fibromyalgia are: widespread pain of at least 3 months' duration and tenderness at

any 11 of 18 defined musculoskeletal sites, referred to as tender points.³ Fibromyalgia may co-exist with any other clinical disorder, so that "a diagnosis of fibromyalgia remains a valid construct irrespective of other diagnoses".³ It may be argued that this is not exceptional, since a diagnosis of Sjögren's syndrome, for example, is not incompatible with rheumatoid arthritis. But, unlike fibromyalgia, these clinical entities have a defined pathology and pathophysiology. Proponents of fibromyalgia seek to diagnose a state of pain, not by invoking any concept of pathophysiology of pain or tenderness, but by finding only the requisite number of tender points.³

The implication that this criterion allows fibromyalgia to be differentiated from other conditions evokes three challenges. First, the false-positive rate of the tender point criterion in the defining study was 19%—ie, 19% of those with 11 of 18 tender points were judged not to have fibromyalgia.³ What condition did they have? Second, how are patients with widespread pain but less than 11 tender points to be diagnosed? Third, what is the correct diagnosis for people with a high tender point count but without widespread pain?

There has been no response to the first challenge.⁴ The second was solved by the decision to use the diagnosis of fibromyalgia for patients with fewer than the required number of tender points, provided that their pain was widespread and that they had many of the so-called characteristic symptoms of the syndrome (fatigue, sleep disturbance, headache, and irritable bowel symptoms).⁸ That was, in effect, a reversion to the 1981 concept.² The third challenge resulted in a fundamental objection to the diagnostic value of tender points, which in the general population are associated with psychological and general distress, independently of the pain status of the individual.¹⁰ Taken together with the recent admission that there is no discrete boundary between fibromyalgia and non-fibromyalgia,¹¹ the diagnostic validity of the tender point count has been refuted. Thus, widespread pain remains the only other criterion for the classification and the diagnosis of fibromyalgia. However, the starting point of the whole exercise of fibromyalgia was the presence of widespread pain. The argument remains irretrievably circular.

The antecedents of fibromyalgia were muscular rheumatism, fibrositis, and neurasthenia—themselves essentialist models of disease based upon speculative ideas of causation.³ To Galenic physicians, the rheuma was a fundamental pathophysiological process, the downward flow through the body of a morbid humour, that caused disease in that part of the body where it came to rest. The term rheumatism was first used in

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Wyllie Arthritis Centre, 17 Lemnos Street, Shenton Park, Western Australia 6008 (J L Quintner MRCP); and **Darlinghurst Arthritis Centre and Pain Research Clinic, St Vincent's Medical Centre, Darlinghurst, New South Wales** (M L Cohen MD)

Correspondence to: Dr John L Quintner

1635 to describe acute arthritis, but by the late 18th century it included painful conditions of non-articular components of the musculoskeletal system.¹² During the 19th century, acute rheumatic fever was delineated, and the various chronic forms of articular rheumatism, including rheumatoid arthritis, osteoarthritis, and gouty arthritis, were distinguished from muscular rheumatism. Rheumatism then denoted either a peculiar type of inflammation within synovium, muscle, fasciae, or the sheaths of nerves,¹³ or, harking back to the concept of rheuma, a widespread bodily disturbance, "(of) not only the blood, and fibrous tissues, but the entire nervous system".¹⁴

When Gowers,¹⁵ in 1904, proposed the term fibrositis, he reasserted the dominant 19th century model of muscular rheumatism by postulating a sterile inflammatory process within muscles that spreads along fascial planes to involve adjacent structures, such as tendons, the sheaths of peripheral nerve trunks, and the capsules of joints. Fibrositis, with its clinical hallmark the "fibrositic nodule", remained a popular diagnosis for (presumed) somatic pain until its putative localised pathological basis was found to be spurious in 1940.⁵ Meanwhile the American neurologist Beard,¹⁶ formulated the concept of neurasthenia in 1869, which literally signified "want of strength in the nerve", to express in more scientific terms the syndrome popularly known as nervous exhaustion. Left untreated, neurasthenia was said to be the forerunner of organic disease. However, as any organic disease could be complicated by neurasthenia, circular reasoning was virtually unavoidable.

The cardinal features of neurasthenia were physical and mental debility, and were frequently grouped together with headache, sleep disturbances, spinal pain and hyperaesthesia, muscular weakness, dyspepsia, and various psychosexual disturbances.¹⁷ Many chronic musculoskeletal pain syndromes (particularly after trauma) that had previously been classified as muscular rheumatism were taken over by neurasthenia.⁵ People with neurasthenia were said to be rarely free from tender spots, the potential foci for widespread pain.¹⁸ Neurasthenia became a convenient somatic diagnosis for many otherwise unexplained symptoms, and bore no stigma of malingering. However, during the 1920s, its popularity waned as theories of aetiology moved away from socially acceptable constitutional factors (such as overwork) towards conscious and unconscious psychological conflicts over sexual disturbances.

People with chronic pain syndromes were then left without a respectable, let alone rational, diagnosis. An insight was claimed by Moldofsky and Scarisbrick¹⁹ who noted the production of neurasthenic symptoms in healthy but sleep-deprived people who led a sedentary lifestyle; they postulated that musculoskeletal symptoms in unfit individuals might be attributable to a disturbance in their sleep physiology.¹⁹ On the basis of this apparent central mechanism, Smythe and Moldofsky²⁰ resurrected fibrositis with the change that tender points no longer signified local pathological change but rather pain amplification within the central nervous system.²⁰

The original intention of these workers was to defend sufferers of chronic pain from accusations of psychogenesis.^{2,21} Individuals with the new fibrositis could be differentiated from healthy people on a

quantitative basis, being more tender at 12 or more of 14 specified anatomical sites than at other non-tender sites.²¹ These tender sites were mostly unknown to the patient and often outside the area of their pain, from which it was argued that the clinical presentation was unlikely to be psychogenic.²¹ Thus, tender points were held to be integral to the diagnosis but incidental to the pain, two propositions that are incompatible.

Fibromyalgia can be seen as a fusion of the still unexplained symptoms of neurasthenia with the localised tenderness of muscular rheumatism and fibrositis. By making it possible to award the diagnosis of fibromyalgia to people with the so-called characteristic symptoms but fewer than 11 tender points,⁸ neurasthenia has been reasserted as a definable clinical entity, but now under a different name.

Because a diagnosis of fibromyalgia depends solely upon clinical features in the absence of any linkage to pathology or pathogenesis, it is not surprising that heterogeneous groups of patients who present with widespread pain and tenderness have been awarded this diagnosis.²² Not only is the use of the diagnosis of fibromyalgia now "out of control",²² but also, as a self-fulfilling disorder of pain modulation, it has become attributed to psychogenic factors.⁶ In effect fibromyalgia, the neurasthenia of the late 20th century, is about to follow its 19th century namesake, paying yet again the penalty of a failure to distinguish between cause and effect.⁹

The clinical phenomenon that those who formulated the concepts of fibrositis/fibromyalgia attempted to understand was excessive musculoskeletal tenderness. Despite intensive research, no peripheral pathological process has been identified in fibromyalgia to explain this phenomenon, which is more accurately termed mechanical allodynia—ie, pain in response to a non-noxious mechanical stimulus.²¹ People who fulfil the criteria for fibromyalgia have mechanical allodynia that is not confined to tender points.^{24,25} Cross-modality psychophysical testing has revealed lowered threshold and tolerance to electrocutaneous stimulation in similar people.²⁶ These observations suggest altered nociception, consistent with the hypothesis of central sensitisation,²⁷ a neurophysiological response to somatic nociceptive input, processed at the dorsal horn of the spinal cord or above. On the other hand, it is argued by some that central sensitisation arises out of ideas, beliefs, emotions, or stress,^{28,29} despite the untestable nature of this proposition. There is evidence that patients with fibromyalgia may be hypervigilant, that is, show an increased tendency to pay attention to bodily events.³⁰ Even so, the question of distinguishing cause and effect remains unaddressed. In this respect fibromyalgia is no different from other entities that describe chronic musculoskeletal pain where pathology in the area of pain complaint is elusive. Although it is not possible to distinguish between altered central nociceptive processing and hypervigilance, the weight of evidence suggests that the pain is more likely to have been caused by afferent neurophysiological events than by psychological factors.

The concept of fibromyalgia has fallen into disrepute because it failed to overcome the essentialist fallacy that was the downfall of its predecessors, muscular rheumatism, neurasthenia, and fibrositis. However, out of the confusion and circular argument, there has

emerged the recognition that clinical mechanical allodynia is the fundamental underlying pathophysiological phenomenon. Whether this condition is due to altered nociception or to altered attention must now be the focus for argument and research.

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