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Editorial

The Lumping/Splitting Conversation Related to Fibromyalgia in Rheumatology: Does It Matter?

Bruce M. Rothschild

Department of Medicine, Indiana University School of Medicine, IU Health, Indianapolis, IN 46202, USA; spondylair@gmail.com

Diagnoses for which there are no pathognomonic laboratory tests are highly dependent on the opinions we call clinical judgement. As such, they are sometimes quite subjective. Should disorders which share some, but not all, manifestations be considered a uniform diagnostic cohort? Support for such an approach seems predicated on the facility with which standard workups and interventions can be pursued, with the effect of such interventions amenable to epidemiological analyses. However, there is a caveat: If the entities included in such a cohort actually differ in their pathophysiology and their response to intervention, such lumping may conceal findings which represent benefits or hazards for one of the components.

While inflammatory arthritis has been a major component of practice, rheumatologists inherited fibromyalgia, not without the still-continuing resistance by many, because of the apparent lack of training/education attention in or interest from other fields of medicine to general body pain and specifically to non-surgical back pain. Such inheritance was not surprising, in view of the position that rheumatologists have held in dealing with clinical situations that often confounded/baffled colleagues in other medical specialties. Frustrated with not only diagnosing but especially with satisfactorily addressing back pain complaints, it was natural to turn to a previously identified source of illumination—the rheumatologist.

Rheumatologists quickly recognized an amalgamation of findings that formed a pattern, to which the name fibromyalgia was ascribed. These include back or generalized pain, sleep disturbance, fatigue, morning stiffness paresthesias, headache, memory compromise and irritable bowel and/or bladder, often accompanied by depression or anxiety [1–3].

However, the issue is a bit more complicated. Fibromyalgia has long been a controversial diagnosis, with significant supporters and deniers of its very existence [4]. Additionally, it has been used as a "waste basket" diagnosis, when the physician could not identify the source of a patient's complaints. Initially diagnosed on the basis of reproducible patterns of trigger/tender points and assurance that tenderness was limited to those sites [5], a subsequent opinion-based approach [6,7] suggested use of the term fibromyalgia for individuals with general or regional body pain. The diagnosis of fibromyalgia has been based on the presence of specific trigger points, in the absence of neutral area tenderness [1,8]. Reproducibility of findings is the important issue. While induced pain or symptom reproduction has a subjective component related to the amount of pressure applied, a semi-quantitative methodology appears effective: Applying sufficient pressure that the examiner's nail bed blanches (turns white).

Allodynia, dysbiosis and general body tenderness are terms used to identify those individuals with tenderness not limited to trigger points, including those who perceive touch sensation as pain. In this spectrum and perhaps an explanation for some instances is [9]. The latter is characterized by stabbing pain, paresthesias, xerophthalmia, altered skin color and sweating patterns. Allodynia has also been viewed as central sensitization syndrome [10–12] or relegated to a somatiform disorder category. The latter is characterized by unexplained severe, intense, disabling, persistent symptoms considered psychopathological [13].

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The rationale for routinely considering all general body pain sufferers as having a single entity, labeling it fibromyalgia [6,7], rather than "dissecting" and segregating cohorts, seems problematic. We have treatment approaches for fibromyalgia that ameliorate symptomatology [14–16]. Those treatments, in my experience, do not work for dysbiosis and only place affected individuals at risk for side effects without likelihood of benefit. Individuals with allodynia, who perceive non-noxious stimuli (e.g., touch sensation) as pain [17–20], do not respond to the interventions that are effective for those individuals with fibromyalgia (diagnosed according to the original criteria) [21].

While complaints of widespread hyperalgesia, fatigue and cognitive challenges do not distinguish among these entities [22], physical examination comes to the rescue. The localized trigger points of fibromyalgia and the general body hyperalgesia of dysbiosis/allodynia seem clearly distinguishable. However, distinguishing fibromyalgia and dysbiosis/allodynia represents the tip of the iceberg. Fibromyalgia also shares symptoms/signs in common with a number of musculoskeletal diseases, with which it is often confused. Polymyalgia rheumatic is one such example, the misdiagnosis of which also places patients at risk of severe side effects from medications which are ineffective in the treatment of fibromyalgia.

The lumper/splitter controversy in recognition of rheumatoid arthritis has seemingly been resolved in favor of the latter by identified biomechanical, biochemical, imaging and epidemiological parameters and studies of the record of inflammatory arthritis in non-humans [23–27]. Characteristics (e.g., joint fusion in the absence of corticosteroid therapy) that are distinctly unusual, if occurring at all, in rheumatoid arthritis facilitate the identification of an individual as not actually having that disease [25–29]. So, too, it is time for reevaluation of our application of the appellation, fibromyalgia.

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