Perspectives

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Can the pain field learn from the functional somatic disorder field?

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"A 41-year-old woman presents at a pain clinic. She developed symptoms 10 years ago, starting with lower back pain, tiredness, chest pain, and palpitations. The pain spread to her joints and muscles with numbness and weakness, and she developed gastrointestinal problems, tinnitus, visual disturbances, and trouble sleeping. Over the past year, severe fatigue developed, causing missed work and weekends spent resting. Her symptoms worsen with stress." If this patient was seen at a pain clinic, the focus would likely be on pain symptoms. However, if she was seen by a cardiologist, the focus would be on heart symptoms, receiving the diagnosis of atypical chest pain, and at the gastroenterologist, she would be diagnosed with irritable bowel syndrome. It is increasingly acknowledged that many chronic pain patients have other distressing symptoms than pain.[4,8,29,35,45,47] While differences exist,[20,46] there is also a significant overlap in the symptoms, predictors, mechanisms, prognosis, and treatment of the patients' various complaints.[4,11,24,28,35]

In this perspective, we argue that when patients experience multiple symptoms in addition to pain, a broader view will strengthen the treatment in the pain clinics as well as research into the underlying mechanisms.

1. Functional somatic disorders

Functional somatic disorders (FSDs) (also called bodily distress syndrome [BDS]) are characterized by persistent and troublesome bodily symptoms that cannot better be explained by other somatic or psychiatric conditions.[4] Patients experience distress due to persistent or recurrent bodily symptoms to the degree that their distress and preoccupation with symptoms interfere with daily functioning often causing repeated contacts with healthcare providers.[4] Functional somatic disorders include different specialty-specific syndromes, including fibromyalgia, irritable bowel syndrome, chronic fatigue syndrome, and multiple chemical sensitivity, but there is often a significant overlap.[8,35,41] In fact, only 19% to 45% of FSD cases have symptoms from one syndrome only, while the remainder have multiple symptoms ascribed to different organ systems (**Fig. 1**).[35] Based on empirical research, a phenotype of multisystem FSD or multiorgan BDS has been identified for patients with symptoms from multiple organ systems.[8,35] At present, no clinical or paraclinical tests that can be used to identify FSD are available. It is established by the presence of a characteristic symptom pattern and the illness picture in general.[4] Like in all diagnostics, all relevant psychiatric and medical differential diagnoses must be considered.

2. Mechanisms of chronic pain in the context of functional somatic disorders

It has recently been suggested that considering fibromyalgia within the concept of FSD may open new avenues for shared underlying mechanisms and treatments,[19] and similarly that the significant overlap in risk factors between chronic primary pain[32] and other somatic symptoms suggests shared mechanisms.[38]

In addition, the focus on mechanisms of fibromyalgia and other idiopathic chronic pain conditions has sometimes been on pain symptoms only, and while acknowledging the presence of other symptoms, these are ignored in mechanistic considerations. For example, in a proposal for clinical criteria and a grading system for nociplastic pain, which has been introduced as a mechanism for patients with unexplained evoked pain, other symptoms such as sleep disturbance and fatigue are considered comorbidities rather than part of the syndrome.[23] It has been suggested that the mechanisms underlying nociplastic pain are amplified processing of or decreased inhibition of pain stimuli,[10] which does not explain other presenting symptoms. The IASP definition of nociplastic pain is "Pain that arises from altered nociception," which may cause confusion as there is no nociception (the neural process of encoding noxious stimuli), and it may lead to a search for peripheral mechanisms. Another term that is often used when discussing some chronic primary pain conditions is central sensitization, which is increased responsiveness to neurons in the central nervous system to their normal or subthreshold afferent input. This term also focuses on the pain system only, and it is much debated as there is no evidence for causality and that it drives chronic pain.[18,42,44] In line with these concerns, altered pain responses measured with pressure pain thresholds and conditioned pain modulation were not seen in a large study of

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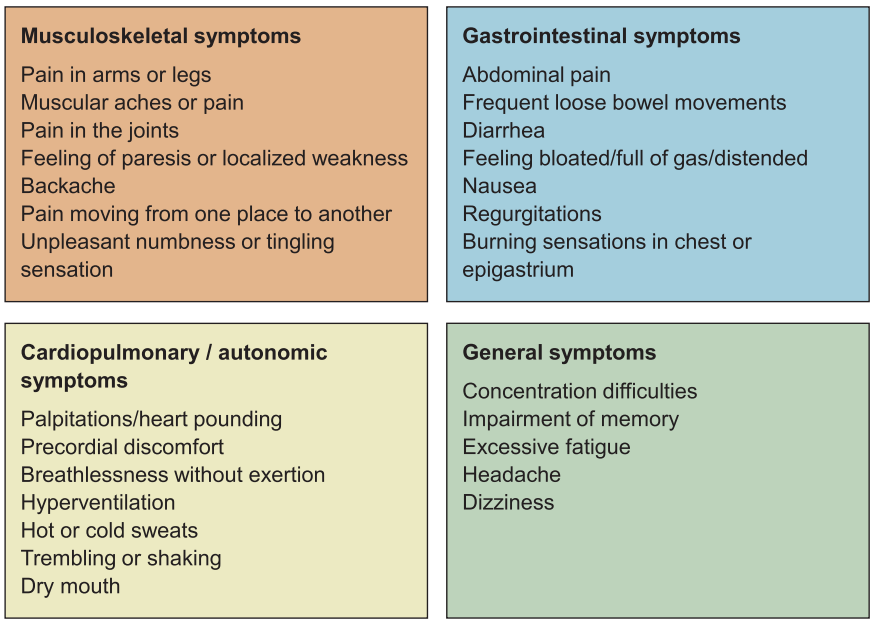

Figure 1. Symptom clusters in functional somatic disorders. Three symptom groups: a musculoskeletal, a cardiopulmonary, and a gastrointestinal group have been identified alongside general symptoms. The groups were identified from a principal component factor analysis of 1000 consecutive patients, and the classification is supported by later studies.[3, 9] A FSD is defined as patients having at least 3 symptoms from one of the 4 groups for at least 6 months, impaired function, and symptoms not better explained by another disease, and multiorgan FSD if patients have symptoms from at least 3 groups.[4]

2198 patients with FSD, including chronic widespread pain and irritable bowel syndrome.[36] The self-report questionnaire the Central Sensitization Inventory is widely used to assess whether central sensitization is the underlying cause of a chronic pain condition despite no scientific evidence to suggest that the questionnaire is a valid indicator of central sensitization.[1] The questionnaire assesses several nonpain symptoms such as fatigue, depression, poor sleep, and lack of concentration. These correlate to psychological measures rather than central sensitization,[1] suggesting that these symptoms should be considered part of the syndrome rather than mechanistic causes of pain. Centrally generated and maintained pain has, on the other hand, been suggested as a term for pain that is independent of nociception or peripheral nerve damage but might be caused by central processing errors, which may cause symptoms other than pain.[25,26]

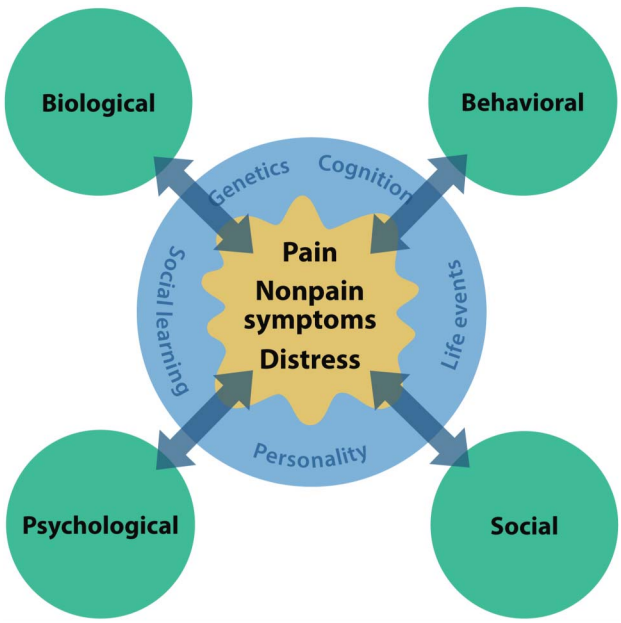

Figure 2. A multifactorial model of chronic pain and functional somatic disorders. The figure illustrates that the development and maintenance of pain and other symptoms (yellow area) depend on patient vulnerability and predisposing psychosocial and biomedical factors (surrounding light blue circle) as well as biological, psychological, social, and behavioral triggering and maintaining factors (light green circles), and similarly that the symptoms may cause biological, psychological, social, and behavioral changes.[27] The arrows indicate bidirectional pathological pathways, which can include cognitive-perceptual and emotional mechanisms, behavioral processes, and disease-specific pathophysiological mechanisms.[27] Future research is needed to understand these predisposing and triggering factors and pathological pathways to improve patient information and treatment.

Functional somatic disorders are believed to have complex, underlying mechanisms, which may vary between individuals.[4] A variety of mechanisms may be involved, including neuroendocrine axis dysregulation, altered interoception, autonomic dysfunction, and immune and metabolic abnormalities. Evidence suggests that many mechanisms are shared across different symptoms including pain,[4,30,34,39] although importantly associations are often found and the cause is not always established.[18] There also seems to be a significant overlap in predisposing factors for FSD, including some chronic primary pain conditions.[32] These include cognitive, environmental, and psychosocial factors such as personality traits, physical and psychological trauma, infections, antibody production, stress, depression, and interaction between expectancies and avoidance.[13–15,17,21,22,31,37,43] We suggest that a focus on pain-specific mechanisms in patients with multiple distressing symptoms, including pain, may not be sufficient and beneficial. Considering postulated mechanisms of chronic primary pain with no known specific cause (eg, small-fiber neuropathy, autoantibodies, or soft tissue lesions) in the context of other distressing symptoms may strengthen research into the underlying mechanisms with larger integration of new scientific knowledge.[4,16,28] Although we do not neglect the possibility of symptom-specific mechanisms (eg, specific pain pathways), we want to emphasize

the need for also including the perspective of common mechanisms contributing to a variety of often co-occurring symptoms. Both chronic pain patients with well-known organic origin and patients with more complex origin where multiple physical symptoms are more often seen can benefit from this approach, as new insights will fertilize both areas.

3. Treatment of chronic pain in the context of functional somatic disorders

In the general population, patients with chronic pain often do not have mental health problems, and many patients with chronic moderate or severe pain do not seek treatment.[2] Patients seen in tertiary pain clinics are often patients who suffer, and suffering and decreased function are important outcomes for treatment success.[5] The treatment of both chronic pain and FSDs often includes patient education, medication, graduated exercise training, and psychotherapy.[5,12,33,40] Using a FSD perspective rather than a narrow pain focus allows addressing all the patients' symptoms. An important mediator of the FSD treatment effect is the patients' illness perception.[6] In FSD treatment, illness perceptions and behaviors are addressed by collaboratively developing an individualized case formulation of the patients' risk factors, precipitating factors, and current perpetuating factors. Patients often find relief in seeing their various symptoms as interconnected and find this holistic approach a key to progress. Furthermore, the treatment challenges the patients' perception of symptoms in general (not only pain symptoms) and thus helps them to cope with their symptoms.

In patients with multiple symptoms (including pain), identifying FSD along with chronic primary pain may prevent patients from receiving different diagnoses for the same health problem. This may prevent the often long odyssey of patients who often visit several specialties and receive different explanations and treatments.[4] It is therefore beneficial to consider all the patients' symptoms simultaneously when treating patients with pain as well as other bothersome symptoms, which is often the case in those with multifocal pain conditions. Patients with known underlying chronic organic diseases can also benefit, independently of treatment options directed at the organic disease component.[27]

We still have limited knowledge of patient-tailored treatment. It is suggested that pain of peripheral and central origin may require different therapeutic strategies,[26] and that patients with local pain may respond differently than patients with widespread pain.[7] Future research is thus needed to identify which patients will benefit from which treatments, including treatments such as patient education and psychological therapy to optimize treatment outcome with fewer resources.

4. Conclusions

We suggest the concept that pain researchers and FSD researchers work together and share their knowledge. Some patients with chronic pain including chronic widespread pain and irritable bowel syndrome frequently report other significant somatic symptoms that also need consideration. We suggest that pain physicians and researchers consider the significance of other somatic complaints and common biological and psychological mechanisms rather than seeing nonpain symptoms only as comorbidities in the treatment of and search for underlying mechanisms when patients present with multiple somatic symptoms. We believe this will facilitate interdisciplinary research into a multifactorial understanding of mechanisms and improving prevention and ultimately a mechanism-based treatment in the pain clinics (**Fig. 2**).

Conflict of interest statement

NBF has received consultancy fees from PharmNovo, NeuroPN, and Saniona, and has undertaken consultancy work for Aarhus University with remunerated work for AKIGAI and Merz.

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