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Toward an Integrative Perspective for Health and Disease

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How does chronic inflammation contribute to depression? Do abnormalities in joint flexibility contribute to anxiety? What are the salutary factors that may prevent or mitigate pathogenesis? These types of general questions gain salience as the models of medical and psychiatric pathologies converge on a final common pathway model [1-3] According to this model, many different factors – genetic, epigenetic, experiential and acquired memories (i.e., information-memes), and stress interact in the balance between health and illness. It is no longer feasible to maintain such dichotomies as functional vs. organic, psychiatric vs. physical syndrome, or even schizophrenia vs. bipolar disorder or anxiety disorder vs. depression.

The three papers in this issue provide good support to this view. Grazziotin et al. state in their review paper of women with chronic pelvic pain that 86% of women with symptomatic endometriosis had co-morbid depression compared to 38% of women with asymptomatic endometriosis [4]. The co-occurrence of depression and pain doubled in economically disadvantaged patients. In other painful pelvic conditions, there was a higher co-morbidity with depression and anxiety. The authors point out that Mast Cells (MC), which are immune cells that mediate inflammation and immunity, contribute to both depression/anxiety and pain through the release of modulators such as cytokines and growth factors as well as direct contact with CNS immune cells, microglia. They indicate that stress, anxiety, and depression are associated with immune dysregulation, which in turn is associated with mast cell activation. They point out that, being located on the brain side of the blood brain barrier; mast cells interact with neurons, glia, blood vessels, and other hematopoietic cells via their neuroactive prestored and newly synthesized mediators. They are first responders, acting as catalysts and recruiters to initiate, amplify, and prolong other immune and nervous responses upon activation. Acute stressors or CNS injury can change both phenotype and numbers of brain MCs. Activation of brain MCs releases neuroactive mediators into the brain parenchyma which may be linked to emotionality. They postulate that the somatosensory system and HPA axis, and their interaction with the immune system, may form a cycle connecting peripheral inflammatory processes and central neuro inflammation that facilitate co-occurrence of chronic pelvic pain and mood disorders.

There is mounting evidence that social support plays an important role in preventing and attenuating the noxious effects of stress and enhancing resilience [5-7]. Theoringer and Woljak developed an animal model of PTSD and evaluated the effect of single vs. dyadic housing on the PTSD-like symptoms they developed following foot shock [8]. They found, however, that in this animal model of PTSD, dyadic housing did not provide the kind of protection expected of social support. This may indicate that social support is much more than simply sharing a residence, especially if that sharing is not by choice.

Sanches et al. studied the possible association between joint hyper flexibility and social anxiety among Brazilian university students [9]. Anxiety and joint hypermobility are known to have some features in common, including early age of onset, higher prevalence in females, lower pain threshold, autonomic alterations, and somatic sensitivity, and have been reported to co-occur significantly. In this study utilizing a structured interview (SCID-IV) to diagnose social anxiety and individual physical examination for joint hypermobility (Beighten Score), there was no significant association between anxiety and joint hypermobility. It is only through exploring possible associations of such syndromes as social anxiety and joint hypermobility that possible underlying mechanisms can be discovered. In this regard, reporting of negative studies is as important as that of positive ones, as they both contribute to the enterprise of elucidating the various pathways to important final common pathway syndromes.

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