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INVITED COMMENTARY

Conceptualizing and Examining the Role of Stress in Arthritis: a Comment on Harris et al

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It has long been known that having arthritis is associated with significant psychiatric comorbidity [1]. For example, approximately 20 to 30 % of patients with arthritis suffer from depressive disorders [2]. It has also been confirmed that having arthritis is a stressful experience for many patients, and that stress may contribute to greater pain, disease activity, and mood disturbance in diagnosed patients [3]. However, while research has documented the negative psychological impact of having arthritis on patients in community samples over time [4], it has never been substantiated that psychological factors, including stress, increase the risk of developing arthritis. This vitally important knowledge would broaden our understanding of the etiology of arthritis and its management. Furthermore, based on evidence that stress may contribute to disease onset, novel treatment strategies could be developed and implemented that could prevent the onset of arthritis, potentially reducing its worldwide prevalence and impact.

While longitudinal studies that are executed on the population level are difficult to design and execute, they provide unique opportunities for examining prospective relationships that can alter our perspective of an entire field. The study by Harris et al. [5] is noteworthy for its magnitude and the significance of its findings. The authors reported results suggesting that perceived stress may have an independent role in the development of arthritis. Using data from a sample of over 12,000 participants from the Australian Longitudinal Study of Women's Health assessed at 3-year intervals, the authors found that perceived stress significantly increased the odds of having arthritis over time. Their multivariate modeling approach, which included a time lag, confirmed the effect of stress on the onset of arthritis 3 years later, while controlling for the contribution of other psychological factors, behavioral variables, disease comorbidities, and health care use. Interestingly, social support, a potentially mitigating factor as a stress buffer, did not protect patients from the onset of arthritis. Nevertheless, greater social support is associated with better mental health functioning in patients who have been diagnosed with arthritis and face the challenges of coping with their illness [6].

Their data also demonstrated that stressful life events were not predictive of arthritis onset. The results illustrate the importance of distinguishing between objective stressors from the feeling of being stressed, showing that the latter is more prognostic of the onset of arthritis. While the objective and subjective components of the stress process have been long debated and studied in the behavioral medicine literature [7], this finding may have particular relevance to autoimmune disease. Subjective stress is correlated with depression and anxiety that drive the sympathetic, neuroendocrine, and immune systems that, in turn, can exacerbate the inflammatory response. Hypothetically, chronic stress might increase the propensity for developing arthritis in persons with genetic vulnerability for autoimmune disorders. Whether this is true or not warrants empirical scrutiny. On a more practical note, it is important to identify patients who are stressed in clinical practice who may be at risk for arthritic conditions. Clinicians can easily assess perceived stress in medical settings by querying patients about their perceived burdens and/or by administering brief self-report instruments [8].

In addition, the Harris et al. study raises important questions about the distinction between osteoarthritis (OA) and rheumatoid arthritis (RA), and whether their data are applicable to either or both conditions. Although their survey

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methodology did not discriminate between these conditions, due to the much higher prevalence rate of OA in individuals over 50 years of age, it is likely that most of their participants had OA. While pain is the central symptom in both conditions, OA and RA differ with respect to etiology, underlying mechanisms, and risk. Obesity, joint trauma, and occupationally based repetitive joint loading are known risk factors for OA. While inflammation historically has not been considered a central factor in the development of OA, joint swelling is a feature of OA reflecting synovitis (local inflammation) [9]. In contrast, RA is an autoimmune, inflammatory disorder that has pervasive, systemic effects. As such, RA would be expected to be more susceptible than OA to perturbations in the hypothalamic/pituitary axis and immune system due to chronic stress that have been shown to affect the inflammatory response. The Harris et al. findings, however, suggest that there may be common psychological and/or biobehavioral pathways linking chronic stress to disease onset in both conditions. The next phase in this research should distinguish between these conditions and address whether perceived stress has a similar or different impact for OA and RA. Future research, using a causal modeling approach that addresses the direct and indirect pathways linking perceived stress to disease onset in both conditions, should be explored.

Finally, this research illustrates the importance of epidemiology to the field of behavioral medicine. Population-based studies have the advantage of examining hypotheses that have widespread significance for the field as a whole that cannot be meaningfully addressed in clinical samples. Most of the studies on the role of psychological factors in rheumatoid research have been conducted with clinic patients and, therefore, have been limited in their generalizability and public health significance. Importantly, the Harris et al. study exemplified the importance of stress in the genesis of arthritis, on the population level, a finding that has the potential for enlightening the field of rheumatology and the care of patients who may be at risk for arthritis-related illnesses. It has established the groundwork for further research that addresses the mechanisms that explain the relationship between perceived stress and arthritis onset.

Conflict of Interest The authors have no conflicts of interest to report.

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