Dear Editor:

I read with great interest the thought-provoking study by Glass et al. [1]. The authors found that, among regularly exercising healthy individuals, some developed chronic fatigue syndrome (CFS) and fibromyalgia (FM). It challenges current views that stress system dysfunction may antedate that stress system dysfunction may stimulate the development of symptoms characterising chronic fatigue syndrome (CFS) and fibromyalgia (FM). It challenges current views tending to reduce the etiological role of stress system impairment in CFS/FM to an epiphenomenon, for example caused by deconditioning or sleep disturbances [2]. Moreover, the findings of this study are in accordance with most CFS/FM patients’ illness history and premorbid lifestyle [3].

Trying to interpret their findings, the authors speculate that those who became symptomatic probably needed to “stimulate” their hypoactive stress system (e.g., by regular physical exercises) to suppress symptoms. Although such an explanation seems plausible at first sight, the reality may be more complex. An alternative hypothesis may place the authors’ findings in a broader psychodynamic/psychobiological perspective.

In our clinical practice, we often hear from CFS/FM patients that—before they got ill—they never could sit still, “they were always busy to avoid becoming nervous”, or “they needed physical workout to chase away negative thoughts or feelings”. In our subsequent research, we were able to objectify the “premorbid overactive lifestyle” of these persons and delineate its underlying meanings [4].

Against this background, we would hypothesise that these persons’ primary need might not be to stimulate their stress system, but rather to regulate it. In other words: to keep their stress system in balance or—to use McEwen’s famous concept—to preserve allostatic balance [5].

But why do some people need (over)activity as a means of stress regulation? To answer this question, we would refer to the fact that an important subgroup of CFS/FM patients reports (more or less severe) childhood adversities [6]. Animal as well as human research has convincingly shown that such experiences may make the stress system more vulnerable [7], influencing later health via physiological, emotional, cognitive, behavioural as well as social pathways [8]. Trauma and abuse usually leads to HPA axis hyperresponsiveness, but hyporesponsiveness may occur as well [9,10]. In this context, physical (over)activity—e.g., by doing a lot of sports—could regulate anxiety, arousal and tension, distract from painful memories and feelings and strengthen self-worth, all contributing to allostatics [2,4].

What happens, then, when an protracted illness or physical injury makes further (over)activity impossible? Such events may seriously threaten the patient’s psychobiological equilibrium and precipitate a dysregulation of the stress system. Although the exact mechanisms of this “allostatic imbalance” are not well understood, three possible consequences may be considered: First, escalating neurohormonal hyperfunction may eventually lead to melancholic depression; second, long-lasting hypofunction may end up in atypical depression, often accompanied by diffuse pain and fatigue [11]; third, after a period of chronic stress (or melancholic depression), the stress system may “switch” from hyper- to hyporesponsiveness via changes in autoregulatory feedback mechanisms, giving rise to a typical fatigue–pain–low mood symptom cluster [12].

Taken together, the findings of Glass et al. [1], especially when interpreted within the above-described psychodynamic/psychobiological perspective, underscore the potential role of (over)activity in the etiopathogenesis of various stress-related disorders. Therefore, this lifestyle factor deserves more research attention, particularly with regard to “unexplained” chronic pain and fatigue [4,13].

References


I. Glass et al. [1], the HPA axis and the genesis of chronic fatigue syndrome.


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