



Journal of Psychosomatic Research 58 (2005) 389-390

Letter to the Editor

Premorbid "overactive" lifestyle and stress-related pain/fatigue syndromes

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 fatigue, musculoskeletal pain and mood changes after a brief period of exercise deprivation, while others remained asymptomatic; they also found that symptomatic subjects were characterised by lower HPA axis, autonomic and immune function (NK cell responsiveness) prior to exercise cessation, suggesting that these subjects had a preexisting hypoactive stress system.

This study is important because it supports the hypothesis that stress system dysfunction may *antedate* 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 *allostasis* [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 *allostasis* [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

- [1] Glass JM, Lyden AK, Petzke F, Stein P, Whalen G, Ambrose K, Chrousos G, Clauw D. The effect of brief exercise cessation on pain, fatigue, and mood symptom development in healthy, fit individuals. J Psychosom Res 2004;57:391–8.
- [2] Cleare A. The HPA axis and the genesis of chronic fatigue syndrome. Trends Endocrinol Metab 2004;15:55–9.
- [3] Van Houdenhove B. Listening to CFS. Why we should pay more attention to the story of the patient. J Psychosom Res 2002;52:495-9.

- [4] Van Houdenhove B, Neerinckx E, Onghena P, Lysens R, Vertommen H. Premorbid "overactive" lifestyle in chronic fatigue syndrome and fibromyalgia. An etiological factor or proof of good citizenship? J Psychosom Res 2001;51:571-6.
- [5] McEwen BS. Protecting and damaging effects of stress mediators. N Engl J Med 1998;338:171-9.
- [6] Van Houdenhove B, Neerinckx E, Lysens R, Vertommen H, Van Houdenhove L, Onghena P, Westhovens R, D'Hooghe MB. Victimization in chronic fatigue syndrome and fibromyalgia in tertiary care: a controlled study on prevalence and characteristics. Psychosomatics 2001;42:21–8.
- [7] McEwen BS. Early life influences on life-long patterns of behavior and health. Ment Retard Dev Disabil Res Rev 2003;9:149–54.
- [8] Kendall-Tackett KA. The health effects of childhood abuse: four pathways by which abuse can influence health. Child Abuse Negl 2002;6/7:715-30.
- [9] Rinne T, de Kloet ER, Wouters L, Goekoop JG, DeRijk RH, van den Brink W. Hyperresponsiveness of hypothalamic – pituitary – adrenal axis to combined dexamethasone/corticotropin-releasing hormone challenge in female borderline personality disorder subjects with a history of sustained childhood abuse. Biol Psychiatry 2002;52: 1102–12.

- [10] Gunnar MR, Vazquez DM. Low cortisol and a flattening of expected daytime rhythm: potential indices of risk in human development. Dev Psychopathol 2001;13:515–38.
- [11] Gold PW, Chrousos GP. Organization of the stress system and its dysregulation in melancholic and atypical depression: high vs low CRH/NE states. Mol Psychiatry 2002;7:254-75.
- [12] Van Houdenhove B, Egle UT. Fibromyalgia: a stress disorder? Piecing the biopsychosocial puzzle together. Psychother Psychosom 2004;73:267-75.
- [13] Vlaeyen JW, Morley S. Active despite pain: the putative role of stoprules and current mood. Pain 2004;110:512-6.

Boudewijn Van Houdenhove

Department of Liaison Psychiatry
University Hospital Gasthuisberg
K.U. Leuven, B-3000 Leuven, Belgium
E-mail address:
boudewijn.vanhoudenhove@uz.kuleuven.ac.be