Not in the mind of neurasthenic lazybones but in the cell nucleus: patients with chronic fatigue syndrome have increased production of nuclear factor kappa beta.

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BACKGROUND: There is now some evidence that chronic fatigue syndrome is accompanied by an activation of the inflammatory response system and by increased oxidative and nitrosative stress. Nuclear factor kappa beta (NFκB) is the major upstream, intracellular mechanism which regulates inflammatory and oxidative stress mediators.

OBJECTIVE AND METHODS: In order to examine the role of NFκB in the pathophysiology of CFS, this study examines the production of NFκB p50 in unstimulated, 10 ng/mL TNF-alpha (tumor necrosis factor alpha) and 50 ng/mL PMA (phorbolmyristate acetate) stimulated peripheral blood lymphocytes of 18 unmedicated patients with CFS and 18 age-sex matched controls.

RESULTS: The unstimulated (F=19.4, df=1/34, p=0.0002), TNF-alpha-(F=14.0, df=1/34, p=0.0009) and PMA-(F=7.9, df=1/34, p=0.008) stimulated production of NFκB were significantly higher in CFS patients than in controls. There were significant and positive correlations between the production of NFκB and the severity of illness as measured with the FibroFatigue scale and with symptoms, such as aches and pain, muscular tension, fatigue, irritability, sadness, and the subjective feeling of infection.

CONCLUSION: The results show that an intracellular inflammatory response in the white blood cells plays an important role in the pathophysiology of CFS and that previous findings on increased oxidative stress and inflammation in CFS may be attributed to an increased production of NFκB. The results suggest that the symptoms of CFS, such as fatigue, muscular tension, depressive symptoms and the feeling of infection reflect a genuine inflammatory response in those patients. It is suggested that CFS patients should be treated with antioxidants, which inhibit the production of NFκB, such as curcumin, N-Acetyl-Cysteine, quercitin, silimarina, lipoic acid and omega-3 fatty acids.

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