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Inflammatory/stress feedback dysregulation in women with fibromyalgia.

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Abstract

OBJECTIVE: Although one of the current hypotheses of the aetiology of **fibromyalgia** (FM) syndrome involves inflammatory and neuroendocrine disorders, its biophysiology still remains unclear. The purpose of the present investigation was to study the systemic inflammatory and **stress** responses, as well as the innate response mediated by monocytes and neutrophils in FM patients.

METHODS: Twenty-five women diagnosed with primary FM and 20 age-matched healthy women (control group) were enrolled in the study. Circulating 'neuroendocrine-**stress**' biomarkers (CRH, ACTH, cortisol, NA, eHsp72, serotonin and IGF-1) were evaluated by ELISA. Serum IL-8 and CRP concentrations were also determined by ELISA, and inflammatory cytokine release by monocytes [IL-1β, TNFα, IL-6, IL-10, IL-18, monocyte chemotactic protein-1 (MCP-1) and RANTES] was evaluated by the Luminex BioPlex system. The phagocytic process of neutrophils (chemotaxis, phagocytosis and microbicide capacity) was also evaluated.

RESULTS: FM patients showed an inflammatory state accompanied by an altered **stress** response. This is mainly manifested by high circulating levels of IL-8 and CRP (in 100% of the FM group), high circulating levels of cortisol, and increased systemic levels of NA and eHsp72. There is also increased release of inflammatory cytokines (IL-1β, TNFα, IL-6, IL-10, IL-18 and MCP-1) by monocytes, and enhanced activation of the functional capacity of neutrophils (chemotactic, phagocytic and fungicidal activities).

CONCLUSION: An inflammatory/**stress** feedback dysregulation underlies FM. Whether dysregulation of the **stress** response is the cause of the inflammatory dysregulation or vice versa is also discussed.

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