

Immune System Activation in CFS

Interleukin-1 beta, interleukin-1 receptor antagonist,
and soluble interleukin-1 receptor type II secretion in chronic fatigue syndrome

Journal of Clinical Immunology 17 (3): 253-261 (May 1997)

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Abstract:

Chronic fatigue syndrome is a condition that affects women in disproportionate numbers, and that is often exacerbated in the premenstrual period and following physical exertion. The signs and symptoms, which include fatigue, myalgia, and low-grade fever, are similar to those experienced by patients infused with cytokines such as interleukin-1. The present study was carried out to test the hypotheses that (1) cellular secretion of interleukin-1 beta (IL-1 beta), interleukin-1 receptor antagonist (IL-1Ra), and soluble interleukin-1 receptor type II (IL-1sRII) is abnormal in female CFS patients compared to age- and activity-matched controls; (2) that these abnormalities may be evident only at certain times in the menstrual cycle; and (3) that physical exertion (stepping up and down on a platform for 15 min) may accentuate differences between these groups.

Isolated peripheral blood mononuclear cells from healthy women, but not CFS patients, exhibited significant menstrual cycle-related differences in IL-1 beta secretion that were related to estradiol and progesterone levels ($R^2 = 0.65$, $P < 0.01$). IL-1Ra secretion for CFS patients was twofold higher than controls during the follicular phase ($P = 0.023$), but luteal-phase levels were similar between groups. In both phases of the menstrual cycle, IL-1sRII release was significantly higher for CFS patients compared to controls ($P = 0.002$). The only changes that might be attributable to exertion occurred in the control subjects during the follicular phase, who exhibited an increase in IL-1 beta secretion 48 hr after the stress ($P = 0.020$). These results suggest that an abnormality exists in IL-1 beta secretion in CFS patients that may be related to altered sensitivity to estradiol and progesterone. Furthermore, the increased release of IL-1Ra and sIL-1RII by cells from CFS patients is consistent with the hypothesis that CFS is associated with chronic, low-level activation of the immune system.