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Cytokines and chronic fatigue syndrome.

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Chronic fatigue syndrome (CFS) patients show evidence of immune activation, as demonstrated by increased numbers of activated T lymphocytes, including cytotoxic T cells, as well as elevated levels of circulating cytokines. Nevertheless, immune cell function of CFS patients is poor, with low natural killer cell cytotoxicity (NKCC), poor lymphocyte response to mitogens in culture, and frequent immunoglobulin deficiencies, most often IgG1 and IgG3. Immune dysfunction in CFS, with predominance of so-called T-helper type 2 and proinflammatory cytokines, can be episodic and associated with either cause or effect of the physiological and psychological function derangement and/or activation of latent viruses or other pathogens. The interplay of these factors can account for the perpetuation of disease with remission/exacerbation cycles. A T-helper type 2 predominance has been seen among Gulf War syndrome patients and this feature may also be present in other related disorders, such as multiple chemical sensitivity. Therapeutic intervention aimed at induction of a more favorable cytokine expression pattern and immune status appears promising.

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