

The Cellular Immunodeficiency Associated with Post-Traumatic Stress Disorder May Be the Result of Sympathetic Overactivity and Be Correctable by Beta-2-Blockers

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Two distinct populations of T cells (TH1 and TH2) have been found mostly to oppose each other, secreting soluble cytokines that regulate cytotoxicity, antibody production, and macrophage function. TH1 cells mainly mediate cellular immunity (through IL-1, IL-12, and IFN-gamma) and TH2 cells preferentially mediate humoral immunity (through IL-4, IL-5, IL-10, and IL-13) [1].

Exposure to trauma can result in immune dysregulation, and increasing evidence suggests the presence of immune alterations associated with post-traumatic stress disorder (PTSD) [2].

Although there are methodological and sample composition differences among the immune studies to date in PTSD, the overall findings support the hypothesis of immune deviation in PTSD toward the TH2 pathway, resulting in suppression of cellular immunity [2, 3]. How the immune dysregulation of PTSD could be explained?

Considerable data indicate a state of hyperarousal in persons who are affected with posttraumatic stress disorder. Patients with this disorder tend to have increased sympathetic nervous system baseline activity and reactivity to stimulation. Some research reports have also found increased urinary excretion of norepinephrine [4].

It has been shown that lymphocytes and macrophages express beta-2-adrenoceptors and that stimulation of these receptors inhibits cellular immune responses [2]. This explains the improvement of cell-mediated symptoms (e.g., arthritis) of systemic lupus erythematosus patients during confrontation with stressors [2]. Furthermore, the cellular immunodeficiency associated with hemorrhagic shock is believed to be caused by increased catecholamine release and is reversible by the administration of beta-blockers [5].

Therefore, it could be concluded that increased catecholamine release in PTSD may, at least partly, be responsible for suppression of cellular immunity in this condition and thus beta-2-blockers may be helpful in correction of this immune dysregulation.

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