

EDITORIAL

Psychological modulation of immune function is now a well-established phenomenon. Psychoneuroimmunology (PNI) provides a distinct perspective regarding the interrelatedness of the nervous, endocrine, and immune systems. PNI explicates the possible means by which behavior and emotion can influence immune function. It embraces the scientific evidence of research with that of endocrinology, neurology and immunology, which shows that the brain and body communicate with each other in a multidirectional flow of information that consists of hormones, neurotransmitters/neuropeptides, and cytokines. Moreover, PNI explains the means by which the immune system affects the nervous system and affects psychological response. The interactions among these systems are mediated at the molecular level by cytokines and hormones produced by cells of not just the immune but also the nervous and endocrine systems. These cytokines and hormones affect endocrine and neuronal processes that, in turn, affect mood, emotions, personal perception, as well as the immune response. Recently, Robert Ader has stated that immunoregulation is not autonomous. This may substantially change the concept of immunology.

Psychoneuroimmunology is an integrative paradigm for advancing both theoretical and empirical knowledge of physiological patterns that contribute to the dynamics of health. Advances in mind-body medicine research together with healthy nutrition and lifestyle choices can have a significant impact on health maintenance and disease prevention. Furthermore, studies of interactions between the nervous and immune systems that effect immunological and behavioral changes are also relevant to our understanding biological issues pertinent to evolution, etiology, ecology, and aging. It is known that changes in maternal hormonal and immune function as a result of stress may adversely affect the immune function and neurodevelopment of the fetus. Recent data suggest that stress induced alterations in gastrointestinal inflammation may be mediated through changes in hypothalamic-pituitary-adrenal (HPA) axis function and alterations in bacterial-mucosal interactions, and via mucosal mast cells and mediators such as corticotrophin releasing factor (CRF). There is evidence that psychologic stress constitutes an increased risk for atopy and influences the disease's clinical course. This risk is believed mediated by the effects of stress on neuroimmunoregulation, which in turn modulates the hypersensitivity response and involves immunoglobulin E-mediated inflammation, helper T-cell 2 predominance, and eosinophilia.

The Editors