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Autoimmune disorders: An overview of molecular and cellular basis in today's perspective

Sayantan Ray, Nikhil Sonthalia, Supratip Kundu and Satyabrata Ganguly
Department of Medicine, Medical College and Hospital, India

Autoimmunity arises when immune responses mounted in the host are directed against self-components. Autoimmune diseases are pathophysiological states that result from a loss of self-tolerance and the consequent immune destruction of host tissues. Autoimmunity is mediated by a variety of molecular and cellular events, and responses. The development of an autoimmune disease is a very complex process in which recognition of selfantigens by lymphocytes is centrally involved in pathologic organ damage. Autoimmune disease is inherited as a complex trait, with multiple loci controlling various aspects of disease susceptibility. More recently, some of these susceptibility genes have been identified. Certain environmental influences, such as cigarette smoke, ultraviolet light, or infectious agents, may interplay with this genetic predisposition to initiate the disease process. Silica exposure and its role in systemic lupus erythematosus (SLE) have been identified in studies of occupational exposure, and experimental studies have explored potential mechanisms related to immune dysregulation. Some autoimmune responses emerge following infection by a pathogen, whose protein(s) hold structural similarities to regions on proteins of the host. Thus, antibodies evoked against a pathogen might cross-react with a self-protein and act as autoantibodies, and the concerned autoantigen then provides a source for persistent stimulation. Evidence is emerging that activation of autoimmune B cells and T cells can be influenced by innate immune receptors, such as Toll-like receptors, which primarily recognize pathogen-derived molecular structures but may cross-react with host molecules. Proteins to which the immune system is generally self-tolerant might, if altered, elicit autoimmune responses. Potential involvement of chaperones in the induction of autoimmune disease pathogenesis has also been explored. The contributions of microRNA to pathogenesis of autoimmune diseases like SLE are beginning to be uncovered and may provide us a new arena for exploration of mechanisms responsible for initiation and pathogenesis of autoimmune diseases.

sayantan.ray30@gmail.com