

Introduction

The Significance of Autoantibodies in Autoimmune Diseases

Autoimmune diseases may be classified classically as organ specific (e.g., Hashimoto's thyroiditis) and nonorgan specific (e.g., systemic lupus erythematosus [SLE]). An alternative classification entails diseases in which the damage is cell mediated (e.g., diabetes mellitus type I) or humoral (autoantibody) mediated (e.g., ITP, autoimmune hemolytic anemia, or myasthenia gravis). In the latter group of diseases, the pathogenetic mechanism is well known and the pathogenic roles of the antiplatelet, anti-erythrocyte, and anti-acetylcholine receptor antibodies are clear cut. Yet many enigmas still exist in our understanding of the immune role of autoantibodies. They include, among others, the fact that in a large number of apparently classical autoimmune disease there are *highly specific* autoantibodies, the targets of which were clearly defined, yet their pathogenic role is still unraveled. Moreover, their existence may be just an epiphenomenon.

In the current issue of the *Critical Reviews in Allergy*, we have decided to concentrate on a series of diseases in which specific autoantibodies are reported. In each one of them extensive research was done to explain their involvement in induction of the clinical manifestations. We are still perplexed how an antibody against topoisomerase I can induce hardening of the skin, how anti-DNA antibody causes central nervous system involvement in SLE, how anti-Ro causes dryness of the eyes, how antiendothelial cells exist in the various vasculitis conditions, how anticardiolipin antibodies are associated with fetal loss and low fecundity rate, and how anticytoplasmic antibodies are involved in Wegener's granulomatosis.

In the following reviews, written by top leaders in their respective fields, one will be able to find answers to many of these complex questions. Yet, we will see that there is still much to be investigated and to be answered.

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