

Autoimmune Diseases

Let us interpret the textbook term "autoimmune disease" as a misnomer - this is not a mischievous challenge, but since our immune system scans our body at each moment, recognising it as self, the disease process stands only for failure of such recognition. Should this occur, then the immune system arrives to the fallacy of self being foreign and aggresses it: autoaggressive disease is the term. Another story is the question: what triggers such failure of self recognition? Experimentally, autoimmune diseases can be induced by various means, e.g. mercuric-chloride induction of glomerulonephritis or, a clinical example, alpha methyl dopamin-induced Coombs-positive hemolytic anemia. The genetic factors are underlined by animal experiments, such as with lupus-prone NZB/W mice. HLA-linkage is underlined with DR4 in rheumatoid arthritis and pemphigus vulgaris, DR3 in insulin-dependent diabetes mellitus and B27 in ankylosing spondylitis. Infectious agents, e.g. Coxsackie virus becomes associated with myocarditis in which there are autoantibodies to heart antigens. Other viruses and Bacteria (*Campylobacter jejuni* in Guillain-Barré disease) become associated with postinfectious autoimmune disease, the following events underlying their pathogenicity: infectious agent fixes to the host cell or mimicks autoantigens by expressing partial sequence homology between the microbial antigen and a self-determinant. Tissue injury by the infectious agent might break tolerance to self antigen thus perpetuating injury by an autoaggressive process, or the infectious agent might enhance expression of MHC antigens.

