

Gregory J Tsay, Department of Medicine and Institute of Immunology, Chung Shan Medical University, Taichung, Taiwan

Systemic rheumatic diseases or autoimmune diseases such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and Sjogren's syndrome (SS) are the consequence of an immune response against self-antigens that results in the damage and eventual dysfunction of target organs. Although the triggering event in most of diseases is unknown, both genetic and environmental factors have long been postulated to explain the development of the disease. Among environmental factors, a large number of candidate organisms, both bacterial and viral, have been suspected.

Viruses and autoimmune diseases

Viral infections are involved in the pathogenesis of autoimmune diseases:

Epstein-Barr virus (EBV) and parvovirus B19: RA and SLE

Coxsackie, CB3 and CB4 strain: IDDM, myocarditis

Retroviruses: HTLV-1, human retrovirus-5

Herpes simplex virus type 1(HSV-1): herpes stromal keratitis(HSK)

lymphocytic choriomeningitis virus (LCMV): multiple sclerosis (MS) or experimental autoimmune encephalitis (EAE)

HCV: Sjogren's syndrome

In animal: virus can induce diabetes and MS

Viruses and autoantibodies

Parvovirus B19 (B19), hepatitis C and B virus, and human immunodeficiency virus (HIV) appear to be associated with autoantibodies more commonly than other viruses. High-titer autoantibodies can be found: antinuclear antibody (ANA), anti-double stranded DNA antibody (anti-dsDNA), antineutrophil cytoplasmic antigens (ANCA), and anti-cardiolipin (aCL), Rheumatic factors (RF), anti-smooth muscle antibody

Although antigenic mimicry has been proposed as a major mechanism by which viruses could trigger the development of such diseases, it is not easy to understand how widely different viruses might induce these autoimmune diseases by this sole mechanisms. A common feature of autoimmune diseases is the breakdown of tolerance of self antigens, a consequence of which is the production of antibodies reactive with multiple self proteins. Apoptosis plays an important role in these diseases. Evidence is accumulating that modifications of autoantigens during apoptosis lead to the development of autoantibodies by passing the normal mechanisms of tolerance.

In previous studies, we have demonstrated the association of parvovirus B 19 infection with the production of ANCA and aCL and B19 non-structural protein (NS1) can induce apoptosis through mitochondria cell death pathway in COS-7 cells. Increased expression and secretion of IL-6 in B19 NS1 transfected COS-7 cells was also found. Our results provide a clue that B19 may play a role in the pathogenesis of autoimmune diseases.