INFECTION AND AUTOIMMUNE DISEASE

How Infectious Agents Cause and Prevent Autoimmune Disorders

© Elaine Moore

Infectious microorganisms can induce autoimmune diseases; however, working efficiently, the immune system's response to infection can also prevent autoimmune disease.

Environmental Triggers

Autoimmune diseases are caused by a combination of genetic and environmental factors. That is, autoimmune diseases develop in genetically predisposed individuals when they’re exposed to certain environmental triggers. Several factors, such as stress, are known to induce or cause flares in nearly all of the different autoimmune diseases. Other factors are specific, contributing to disease development and exacerbations (worsening of symptoms) in only certain autoimmune diseases. For instance, silica dust is known to trigger scleroderma.

The Role of Infectious Agents

Infectious agents include viruses, bacteria, parasites, fungi, and other organisms that cause infection. Recent studies show that infectious agents have variable effects on autoimmune disease, with protection from autoimmune disease being a more frequent response to infection than previously thought.

1. Infectious agents can induce autoimmune disease
2. Infectious agents can enhance autoimmune disease
3. Infectious agents can abrogate or offer protection from autoimmune diseases

The Hygiene Hypothesis

Increasing evidence shows that our immune system’s normal response to infection results in an amelioration of autoimmune disease symptoms and protection against autoimmune disease development. This evidence supports the theory known as the hygiene hypothesis, which states that increased use of antibiotics, antibacterial and antiseptic cleaning agents, and vaccines leads to an enhanced incidence of autoimmune disorders, asthma, and allergies.

Infection and Protection

Normally, infection of target cells and organs causes the release of sequestered (normally hidden from the blood circulation) autoantigens (self protein particles). Antigen presentation to immune system cells is thereby enhanced. Although these antigens
normally provoke a heightened inflammatory response, some of these antigens can activate regulatory T-lymphocyte cells, which dampen rather than evoke aggressive immune responses.

The immune system chemicals known as cytokines or chemokines are also released during the immune system’s response to infection. Chemokines can order aggressive T lymphocytes to the site of infection, drawing them from the autoimmune process.

Type 1 diabetes and multiple autoimmune syndrome are induced by Cytomegalovirus (CMV); type 1 diabetes is also associated with rotavirus in children and abrogated or prevented by infection with other organisms that target organs distant from the pancreas.

Autoimmune hepatitis, myasthenia gravis, cryoglobulinemia, and rheumatoid diseases are induced by the hepatitis C virus.

Multiple sclerosis (MS) and systemic lupus erythematosus (SLE) are both induced by the Epstein-Barr virus (EBV). SLE is also induced by human parvovirus 19 and MS is also associated with human herpesvirus 6.

Sjogren’s syndrome and autoimmune myocarditis are both associated with the coxsackievirus strains B3 and B4.

Autoimmune gastritis is associated with Helicobacter pylori, which is also associated with gastric ulcers and stomach cancer.

PANDA syndrome and rheumatic heart disease are both associated with Streptococcal infection.

Graves' disease is associated with Campylobacter jejuni infection and retroviral infections.

Limiting Factors

The precise timing of the immune system response to infection, the particular strain of infectious agent, and the magnitude of inflammation can all influence whether infection induces or prevents infection. Researchers conclude that the entire infectious history of each patient might determine the overall immune status that results in autoimmune disease or not. The role of good nutrition and antioxidants in reducing inflammation and oxidative stress must also be considered.

Resources:

Urs Christen and Matthias von Herrath, Infections and Autoimmunity---Good or Bad, Journal of Immunology, Brief Reviews, 2005, 174: 7481-7486.
Robert Fujinami, Matthias von Herrath, Urs Christen, and J Lindsay Watson, Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease, Clinical Microbiology Reviews, Jan 2006, 19: 80-94.

The copyright of the article Infection and Autoimmune Disease in Autoimmune Disease is owned by Elaine Moore. Permission to republish Infection and Autoimmune Disease in print or online must be granted by the author in writing.