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Environmental Factors in Autoimmune Disease

Autoimmune diseases are chronic, potentially life-threatening conditions. There are more than 80 recognized autoimmune diseases including systemic lupus erythematosus, glomerulonephritis, multiple sclerosis, autoimmune thyroiditis, rheumatoid arthritis, and myositis. Although some of the conditions afflict only small numbers of individuals, as a group autoimmune diseases represent an important public health concern. The common characteristics of these diseases are immune responses directed against healthy tissue or cellular components normally protected from immune attack and the resultant inflammatory response.

Although genetic susceptibility and exposure to infectious agents have been identified as possible contributors to autoimmune disease and have been extensively studied, these factors cannot account for most cases. This suggests the likelihood of exposure to environmental agents as an etiologic factor, and research has linked environmental agents with autoimmunity. Human studies have shown an association with exposure to vinyl chloride, silica, and organic compounds. Likewise, experimental studies have shown numerous autoimmunity-related immunologic

changes induced by exposure to metals, polycyclic aromatic hydrocarbons, and mycotoxins. Possible mechanisms for environmentally induced autoimmunity include molecular mimicry, alteration of lymphocyte signaling, and interference in the development of tolerance to self-antigens.

Despite the accumulation of these research data, there are still gaps in knowledge, including how to link results from human and animal studies. In February 2003, the NIEHS, along with other NIH institutes and centers, cosponsored a workshop titled "Environmental Factors in Autoimmune Disease."



The goals of the workshop were to obtain input from the environmental health science and autoimmune research communities on the most appropriate and productive directions for research in the area of environmentally related autoimmune disease.

The workshop was attended by more than 100 participants, including basic scientists, epidemiologists, clinicians, and disease advocates, and included a session consisting of six breakout groups focused on the following topics: gene—environment interactions, altered antigens, immune modulations, signal transduction, translational research: systemic autoimmune disease, and translational research: organ-specific diseases. The meeting yielded two primary findings. First, although experimental animal data are strong, many more human studies, both epidemiologic and clinical, are needed to link environmental exposure to autoimmune disease. Second, greater efforts are needed to establish collaborations between epidemiologists and clinicians on the one hand, and basic scientists on the other.

The NIEHS is interested in receiving investigator-initiated applications in the area of environmental factors on autoimmunity.

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