autoimmunity, the state in which the immune system reacts against the bodyâ€™s own normal components, producing disease or functional changes.

The human immune system performs a surveillance function, identifying and disposing of antigensâ€”materials such as toxins or infectious microbes that it recognizes as foreign. This surveillance is carried out mostly by the white blood cells called lymphocytes, which recognize foreign antigens and either attack them directly or produce antibodies against them. With a vast diversity of antigen-fighting agents in constant circulation, some are inevitably produced that would react to self antigensâ€”healthy cells or harmless substances of the body that the immune system treats as if they were foreign. Normally, lymphocytes that would trigger immune reactions to the bodyâ€™s own tissues are eliminated before they mature. How this occurs is not completely understood. There is evidence that self-reactive T lymphocytes, or T cells, are killed in the thymus, whereas B lymphocytes, or B cells, that would produce autoantibodies are prevented from maturing after they leave the bone marrow.

For reasons that are little understood, the elimination process sometimes fails, producing autoimmune disorders or diseases. Several mechanisms of failure have been postulated. Chemical, physical, or biological agents may alter self components so that lymphocytes that normally recognize them as safe would then react to them as foreign. Infectious agents may also produce antigens so similar to those on healthy cells that lymphocytes or antibodies would react to both kinds indiscriminately. (This phenomenon is known as cross-reaction.) Self antigens (such as those found in the lens of the eye) that normally do not come into contact with circulating immune agents may, through tissue infection or injury, be brought into contact with them, triggering a response. Suppressor T cells, lymphocytes that restrain the action of antibody-producing B lymphocytes, may somehow cease functioning. There is also evidence that there may be a genetic predisposition to specific autoimmune diseases. The higher incidence of autoimmune disease in women may indicate a sex-linked or hormonal influence. Although the ultimate cause of autoimmune diseases may not be known, the development and course of many autoimmune diseases is now better understood.

Autoimmune attacks follow a variety of routes. In one route, circulating antibodies bind to cells and either assist in destroying them or interfere with their functions. In another route, antibody-antigen combinations circulate in the blood and lymph systems, lodge in various tissues, and cause cell destruction. In yet another route, cell-killing lymphocytes launch a direct attack on healthy tissues.

Autoimmune diseases are divided into two classes: organ-specific and systemic. An organ-specific disease is one in which an immune response is directed toward antigens in a single organ. Examples are Addison disease, in which autoantibodies attack the adrenal cortex, and myasthenia gravis, in which they attack neuromuscular cells. In systemic diseases the immune system attacks self antigens in several organs. Systemic lupus erythematosus, for example, is characterized by inflammation of the skin, joints, and kidneys, among other organs.

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Because the cause of immune system failure is unknown, treatment of autoimmune diseases centres on alleviating symptoms such as inflammation. In organ-specific disorders, attempts are made to correct the specific defect. Drugs that suppress the production of antibodies must be used carefully to avoid lowering the bodyâ€™s resistance to infection.

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