

Molecular Mimicry and Autoimmune Diseases

Although our understanding of the complexities of the immune system continues to grow, most scientists agree that we have much to learn about "normal" immune responses as well as misdirected immune system attacks, generally termed "autoimmune diseases." It is widely accepted that an autoimmune disease begins when the immune system mistakenly identifies the body's own cells as "foreign invaders" (such as viruses or bacteria) and sets about to destroy these cells. Scientists who study the immune system believe that some of the answers about the nature of autoimmune diseases, such as Juvenile Diabetes and Lupus, may lie in the controversial theory of "molecular mimicry." This theory, which may help to explain how autoimmune diseases are triggered, suggests that certain common disease-causing agents (e.g., bacteria or viruses) may initiate a mistaken autoimmune response causing the immune system to attack the body's own tissues as well as the invading offender (the "mimic"). Molecular mimicry does not attempt to explain the role that genetics (i.e., genetic susceptibility) or other environmental or hormonal factors may play in the autoimmune disease process.

Certain specialized cells of the immune system known as T cells (or killer T cells) mature in the thymus and are released into the blood stream where they play a major role in immune functions. These cells are able to "recognize" the body's own cells (self proteins) as well as the proteins of foreign invading agents (antigens). In most cases, during a normal response, the immune system appropriately destroys the invading agent, disregarding the body's own cells.

For example, when a person has a respiratory infection, the virus "makes a home" in the lining of the throat. During a "normal" immune response, specialized "defender" cells (e.g., macrophages or B cells) surround the invading virus, eventually "chopping it up" into tiny protein fragments (peptide chains) that are strung together like a necklace. Some of these fragments eventually make their way to the surface membranes of these specialized immune cells where they park themselves in ridges (clefs) on the cells_in effect, announcing to the body that an invading virus or bacterium is present. Scientists suspect that those fragments that make their way to the surface of the cells may be determined, in part, by our genetic makeup. It is believed that the presence of these protein fragments on the cell's surface evoke a response from the "killer"

T cells of the immune system. Though present by the millions, each T cell is uniquely shaped and different so as to recognize a specific protein fragment. T cells that "match" the protein sequence of the surface fragment "anchor" onto the viral protein. Thus begins the process of destroying the cell that contains the virus.

Scientists believe that this normal immune response may go astray when protein fragments from invading viruses or bacteria "mimic" or are very similar to one of the body's own proteins. The result may be the beginning of an autoimmune response, when

the immune system attacks its own tissues. Essentially, the immune system is fooled and responds inappropriately because it is unable to distinguish between the "mimic" protein on the surface of the "defender" cells and the body's own proteins. Researchers believe that this may be the case in certain diseases such as Juvenile Diabetes. The cells of the pancreas that produce insulin (islet cells) contain a "normal" protein sequence (GAD) that is similar to the protein fragments of a virus, such as the Coxsackie virus. The immune system may target the virus "mimic" and then mistakenly destroy the cells of the pancreas that have a similar protein sequence on the surface of the islet cells.

Many mysteries have yet to unfold as scientists unravel the sequence of events surrounding normal and abnormal immune responses. Many questions remain and skeptics believe that the theory of molecular mimicry requires additional scientific investigation. However, proponents of the theory of mimicry believe that unraveling the triggers of the autoimmune disease process, which genes may be involved, and how specific T cells are activated may very well hold the clues that may lead to new treatments and possible prevention of autoimmune diseases in the future. Much of the important new information that scientists have learned about the immune system was made possible through the study of HIV and AIDS. This knowledge is now becoming applicable to other diseases.