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Perspective

Mechanisms for Preventing Immune Responses against Autoimmunity and Endogenous Antigens

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DESCRIPTION

The immune system is a remarkable defense mechanism that protects the body against harmful invaders such as bacteria, viruses, and parasites. However, to function effectively, the immune system must also distinguish between foreign invaders and the body's own tissues. The mechanisms that prevent immune responses against self-antigens are essential for maintaining immune tolerance and preventing autoimmune diseases. Central tolerance is an important mechanism that operates during the development of immune cells in primary lymphoid organs like the thymus and bone marrow. It ensures that potentially self-reactive immune cells are eliminated or rendered harmless before they mature. In the thymus, developing T cells undergo a process called negative selection. T cells that react too strongly to self-antigens presented by thymic epithelial cells are eliminated through apoptosis. This process helps to remove T cells that could potentially target and harm the body's own tissues. B cells that exhibit a strong affinity for self-antigens in the bone marrow are also eliminated. B cells that survive this selection process often undergo receptor editing, where their antigen receptors are modified to reduce self-reactivity.

While central tolerance mechanisms are powerful, some self-reactive immune cells can escape and reach peripheral tissues. Peripheral tolerance mechanisms act outside primary lymphoid organs to restrain and regulate these self-reactive cells. Self-reactive T cells that encounter self-antigens in peripheral tissues without appropriate co-stimulation become functionally unresponsive or anergic. This prevents them from initiating immune responses even if they encounter their corresponding antigens. Tregs are a specialized subset of T cells that play a crucial role in maintaining immune tolerance. They suppress the activity of other immune cells, including self-reactive T cells, preventing them from attacking self-tissues. Tregs achieve this through the secretion of immunosuppressive molecules and direct cell-to-cell interactions. Dendritic cells in peripheral tissues can promote immune tolerance by inducing a state of tolerance in potentially self-reactive T cells. This is achieved through mechanisms like inducing T cell energy, promoting the generation of Tregs (Regulatory T cells) or inhibiting the production of pro-inflammatory cytokines. Cytokines and regulatory molecules play a pivotal role in maintaining immune balance and preventing excessive responses against self-antigens. Cytokines like Interleukin-10 (IL-10) and Transforming Growth Factor-Beta (TGF-β) have immunosuppressive effects. They inhibit the activation of immune cells and help prevent the development of autoimmune responses. Immune checkpoints are molecules that regulate the activity of immune cells. CTLA-4 (Cytotoxic T-Lymphocyte-Associated Antigen-4) and PD-1 (Programmed Death-1) are examples of immune checkpoint molecules that inhibit the activation of T cells, preventing them from attacking self-tissues. These checkpoints are crucial for preventing excessive immune reactions that could harm the body.

Genetic and epigenetic mechanisms play a role in shaping the immune response to self-antigens. Genes that encode for self-antigens are often turned off or down regulated in thymic epithelial cells. This reduces the likelihood of strong self-reactive T cell responses during negative selection. Epigenetic changes can alter the expression of self-antigen genes in peripheral tissues, influencing their visibility to the immune system. This can help prevent unnecessary immune responses against harmless self-antigens. These mechanisms ensure immune tolerance, maintain tissue integrity, and prevent the development of autoimmune diseases that arise when the immune system targets its own tissues.

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