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Opinion Article

Immunogenetics of Auto Immunity

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DESCRIPTION

Autoimmune diseases such as type 1 diabetes are complex genetic traits that result from defects in the immune system. Identification of genes that define immune defects can identify new target genes for therapeutic approaches. Alternatively, genetic variation can help to define immunological pathways that lead to disease. The term immunogenetics consists of two words, immunology and genetics, and is defined by medical subject headings as "a subfield of genetics dealing with the genetic basis of the immune response (immunity)." The transfer of genes from the parent to the offspring of the organism within the framework of the organism's genes (DNA strands) and possible changes is the basis of genetics.

Immunology deals with the biological and biochemical basis of the body's defense against pathogens (bacteria, viruses, fungi, etc.) but foreign substances such as biological toxins and environmental pollutants, and their defense mechanisms. It also deals with defenses against failure and dysfunction. In addition to these external effects on living organisms, there is also a defensive response to the body's own cells. Body's response to cancer or a part of the body's failure to respond to healthy cells is part of an immune disorder. Therefore, immunology is a subcategory of biology. Its origin is usually due to Edward Jenner, who discovered in 1796 that cowpox elicited protection against human smallpox. The term Immunogenetics includes all processes of an organism that are regulated and influenced by the genes of the organism on the one hand and are important for the immune defense response of the organism on the other. The development of most autoimmune diseases has a strong genetic component. Genetic contributions to the disease range from the simple Mendelian laws of the causative allele to the complex interactions of multiple weak loci that affect risk. Disease-causing genetic variation is discovered through a variety of strategies, from chain studies to genome-wide association studies. Both environmental and genetic triggers are involved in the pathogenesis of autoimmune thyroid disease such as Graves' disease and Hashimoto's thyroiditis. The exact etiology and causality between the environment and genes is unknown, but Graves' disease and Hashimoto's thyroiditis share similar immune disease mechanisms. Both are characterized by thyroid autoantibody production and thyroid lymphocyte infiltration, but are clinically distinct entities with thyrotoxicosis in GD and hypothyroidism in HT. Family and population studies confirm strong genetic impact and heritability in the development of autoimmune thyroid disease.

Autoimmune thyroid disease susceptibility genes can be classified as either thyroid-specific or immunomodulatory. Of the Autoimmune thyroid disease susceptibility genes, FOXP3 and CD25 play important roles in establishing peripheral tolerance, and the CD40, CTLA4 and HLA genes are important for T lymphocyte activation and antigen presentation. In particular, polymorphisms in these immunomodulatory genes contribute significantly to the predisposition to GD, HT and, of course, other autoimmune diseases. Single nucleotide polymorphisms (SNPs) in immunomodulatory genes functionally impede the proper development of central and peripheral tolerance and T cell interactions with Antigen-presenting Cells (APCs) at the immunological synapse. It suggests that it may change. Therefore, autoimmune thyroid disease susceptibility genes directly contribute to the development of organ-specific autoimmunity, an important mechanism underlying the disruption of self-tolerance. The major immune modulating genes that is associated with autoimmune thyroid disease and their potential functional effects on thyroidal immune dysregulation. Since 1972 (histocompatibility and immunogenetics) organizations have been founded specializing in research activities on a large number of different questions in immunogenetics. Both the acceleration of and the decreasing costs for the sequencing of the genes have resulted in more intensive research of both academic and commercial working groups. Current research topics particularly deal with forecasts on the course of diseases and therapy recommendations due to genetic dispositions and how these dispositions can be affected by agents (gene therapy). A special focus is often laid on the forecast regarding and therapy of genetically based autoimmune diseases, which include all diseases caused by an extreme reaction of the immune system

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against the body's own tissue. The immune system mistakenly recognizes the body's own tissues as foreign bodies to fight. The result can be a severe inflammatory response that can cause permanent damage to the affected organ. Autoimmune diseases that develop and/or have a course in the individual genomes of an organism include multiple sclerosis, type I diabetes, rheumatoid arthritis, and Crohn's disease. Similar to multiple sclerosis, autoimmune disease is not caused by genetic variation, but that its course and treatability are greatly influenced by genetic predisposition.