Genes involving immunological pathway of Multiple sclerosis

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Multiple sclerosis (MS) is a chronic autoimmune demyelinating disease of the central nervous system (CNS) characterized by inflammation, demyelination and primary or secondary axonal degeneration. Autoimmune diseases reveal typical characteristics of common complex diseases. These diseases are non-Mendelian, but frequently show familial clustering. Recent data, however, suggest that the pathogenesis of multiple sclerosis is much more complex and that autoimmune mediated inflammation may only be responsible for a part of the disease spectrum. Previous studies have confirmed that the onset of MS is the result of the interplay of environmental factors and genetics. The genetics of common complex diseases, many of which are autoimmune, are difficult to understand. It is clear that the susceptibility to MS, results from interactions of genes, environmental interactions and gene/environment interactions.

T-Cells play a particularly important role in MS, but Damage of CNS, is however, most likely mediated by other components of the immune system, such as antibodies, complement, CD8+ T cells, and factors produced by innate immune cells.

Susceptibility to MS is linked to genes in the MHC (Major Histocompatibility Complex) on chromosome 6. Other genes within the HLA (Human Leukocyte Antigen) complex are involved in the pathogenesis of MS, including TNF-Alpha (Tumor Necrosis Factor-Alpha), various components of the complement cascade, and myelin oligodendroglial glycoprotein. In addition, some other genes such as IL2RA, IL7RA and IL12 have been recognized as associated genes with MS. Given to previous studies, most of genes that are known to take part in susceptibility or pathogenesis of MS, are genes that play important role in immune system.

It is now obvious that MS is not just a disease of the immune system, but that factors contributed by the central nervous system are equally important and must be considered in the future.

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