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Genetics of Autoimmune Diseases

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Introduction

Population genetic forces are responsible for human genetic variation. This genetic diversity affects illness risk and contributes to inequities in health. Autoimmune diseases (ADs) are a group of complicated, varied conditions characterized by immune reactions directed against oneself. ADs are widespread, have gender and ethnic inequities, and are becoming more common. Because natural selection has such a strong influence on human genetic variation and immune function genes are enriched for positive selection signals, it's thought that the prevalence of AD risk alleles seen in different populations is partly due to different selective pressures (such as pathogens). With the development of high-throughput technologies, new analytical approaches, and large-scale studies, evidence for natural selection's role in contributing to heritable traits is becoming more abundant. Population genetics phenomena help explain a lot of the intricacies of gene effects in different ADs. Integrating Alzheimer's disease susceptibility research with population genetics to study how natural selection has influenced genetic variation that determines disease risk will aid in the identification of functional variations and the elucidation of biological mechanisms. Autoimmune diseases (ADs) are a group of more than 80 chronic, frequently disabling illnesses marked by immune system failure, which results in a loss of tolerance to self-antigens, an increase in autoantibodies, inflammatory and mediatory cells, and chronic inflammation. Despite the participation of numerous organ systems, the AD family is notable for its intricacy and comparable underlying mechanisms. Patients frequently experience debilitating symptoms for the rest of their lives, as well as organ loss, decreased productivity at work, and excessive medical costs. Many ADs can have an effect on fetal and maternal outcomes, such as pregnancy loss in women with systemic lupus erythematosus, vasculitis, and type 1 diabetes, and infertility in women with rheumatoid arthritis, as they cheval before or during a woman's reproductive years [1-3].

Description

ADs are widespread, affecting five to nine percent of the population and posing significant personal and societal health risks. The causes of its high frequency, gender and racial differences, as well as their increased incidence and prevalence, are unknown. Disease concordance in monozygotic twins is four times higher than in dizygotic twins in rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), type I diabetes, and multiple sclerosis (MS). In studies of MS, type I diabetes, Graves' disease, discoid lupus, and SLE, strong familial connections were discovered. Multiple susceptibility genes involving MHC and non-MHC regions have been discovered in recent genome wide searches in RA, SLE, and MS; there is also evidence that many autoimmune disorders share a similar set of susceptibility genes. Because genetic risk factors are complex and illness penetrance is limited, environmental factors and gene-environment interactions may play a role in the aetiology of autoimmune disorders. Some research in recent years has looked into the genetic ties to various autoimmune illnesses, particularly organ-specific ones. Some data on the genetic origin of primary biliary cholangitis (PBC), an autoimmune cholestatic liver disease characterized by antimitochondrial autoantibody positivity and an accumulation of antigen-specific autoreactive B and T lymphocytes targeting biliary epithelial cells, is accessible herein. The cause of PBC is unknown; however, like other autoimmune diseases, the human leukocyte antigen (HLA) class II alleles have been found to be strongly linked to disease vulnerability. Non-HLA genes have also been linked to the development of PBC in recent genome-wide association studies [4].

Genetic variants of the IL12A and IL12RB2 genes were shown to have the highest connections, while additional polymorphisms, such as STAT4 and CTLA4, were also discovered. The identification of certain genetic variations may aid in the understanding of pathogenic mechanisms underlying PBC development as well as the identification of patients who have a more aggressive disease history. The significance of virus-derived microRNA in genomic stability is another aspect of the genetics and autoimmune scenario. Infections, as has been proven, are important environmental variables in the development of autoimmune disorders. Viral infections could interact with the host genome via noncoding RNAs called microRNA, allowing alterations that could be responsible for immune response dysregulation, as the Epstein-Barr virus has been documented to do. The focus of genetic research has switched in recent years to other autoimmune illnesses and environmental factors that may alter the host genome. These new insights on the mosaic between genes and diseases may help researchers better understand the pathogenic pathways that lead to the loss of tolerance and the emergence of immune-related diseases [5].

Conclusion

Many risk loci for autoimmune disorders (ADs) have been identified by genome-wide association studies (GWASs), which have provided insight into the aetiology of each disease. Some of these loci are shared among distinct ADs, such as PTPN22, IL23R, and STAT4, and the mix of risk loci may define an individual's susceptibility to the illness. Most ADs, like other complex traits, are multifactorial, involving both genetic and environmental components in its origin. Individual and population differences influence the genetic basis of disease. Mutation, migration, genetic drift, and natural selection at the population level have all left their mark on genetic variation, which is likely to influence phenotypic expression in specific populations.

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