Genotype polymorphism of *Mycobacterium leprae*

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EDITORIAL

Mycobacterium leprae, commonly known as Hansen's disease, causes leprosy, a chronic infectious illness caused by a bacterium that prefers the skin and nerves. One or more of the three cardinal signs: hypopigmented or erythematous skin patches with definite loss of feeling, thicker peripheral nerves, and acid-fast bacilli found on skin smears or biopsy material describe the condition clinically. *M. leprae* mainly infects Schwann cells in the peripheral nervous system, causing nerve damage and disability. Even though the incidence of *M. leprae* infection has decreased in endemic countries since the World Health Organization (WHO) implemented a multidrug treatment (MDT) programmer to treat leprosy, new case detection rates remain high, indicating ongoing transmission. The host immune response is blamed for mycobacteria susceptibility and the illness's clinical history.

Mycobacterium leprae

M. leprae is a significant human pathogen that is an acid-fast bacillus. Leprosy has been found in nine-banded armadillos and three primates, in addition to humans. The bacteria may also be cultivated in the lab by injecting it into mice's footpads. Mycobacteria are infamous for their sluggish development. *M. leprae* has yet to be effectively grown in vitro due to its 14-day doubling period. It has a coding capability of less than half and a significant number of pseudogenes. The remaining *M. leprae* genes aid in defining the minimal gene set required for this mycobacterial pathogen's in vivo survival, as well as genes that may be essential for infection and pathogenesis in leprosy. *M. lepromatosis* is a newly discovered mycobacterium that is known to cause disseminated leprosy, but its relevance is unknown.

Host response to genetic determinants

Human genetic variables have a role in leprosy transmission and disease progression. A low Lymphotoxin-Producing Allele (LTA) was found to be a substantial genetic risk factor for early onset leprosy in Single-Nucleotide Polymorphism (SNP) association studies. Other SNPs linked to illness and/or the development of responses in genes including vitamin D receptor (VDR), TNF-, IL-10, IFN-, HLA genes, and TLR1 have also been proposed. Linkage studies have shown polymorphic risk factors in the promoter region of two genes: PARK2, which codes for Parkin, an E3-ubiquitin ligase, and PACRG. According to research, NOD2 genetic variations are linked to leprosy susceptibility and the development of responses (type I and type II). By regulating the interaction between people and viruses, genetics plays a critical role in determining susceptibility to infectious illnesses. This is especially true in the case of leprosy, where the etiological agent, *Mycobacterium leprae*, has semi clonal features that are incompatible with the vast range of disease presentations. Several gene variations have been identified as risk factors for leprosy in general, illness clinical manifestations, and the incidence of leprosy responses in recent decades as a result of genetic investigations. Several of these genes are immune-related, as predicted; nevertheless, hypothesis-free techniques have led to genes that are not traditionally associated with immune response. PARK2, which was first identified as a Parkinson's disease gene. Even with robust, hypothetical study models such as genome-wide association analyses, most of the impact of key genes determining leprosy susceptibility remains uncertain.

New genes and potential pathways that contribute to disease pathogenesis have been found as a result of research on the host genetic component of leprosy. Many of the findings were validated in ethnically diverse groups, indicating the critical relevance of the pathways identified by host genetic research for leprosy susceptibility. Despite advances in understanding the role of host genetic variations in leprosy development, a complete picture has yet to emerge. Recent research has found that the genetic contribution to leprosy susceptibility varies between children/adolescents and adults, for example. Furthermore, little is known about the influence of epigenetic mechanisms and uncommon genetic variations on primary protein structure and biological function in leprosy.

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