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Short Communication

Genetic Inheriatance to Genomic Sequencing Patterns of Leprosy

Jeremy Walton*

Department of Medicine, University Paris-Sud, Orsay, France

ABOUT THE STUDY

Leprosy, also known as Hansen's disease (HD), is an ancient bacterial infection that, while being treatable, is a major public health issue in many areas of the world. HD is caused by infection with the *Mycobacterium leprae* bacillus, which causes a long-term infection in humans that mostly affects peripheral nerves and skin but can also damage the eyes, mucous membranes, bones, and testes, resulting in a wide range of clinical phenotypes. *Mycobacterium leprae* prefers keratinocytes, macrophages, and histiocytes in the skin. Schwann cells in peripheral nerves contain *Mycobacterium leprae*. In response to *Mycobacterium leprae* antigens, keratinocytes appear to play a major role in the production of the antimicrobial peptide defensing. The disease's dermatological signs are caused by bacilli in the skin, and nerve infection causes axonal malfunction and demyelination, which leads to sensory loss and its implications of disability and deformity. In this regard, the degenerative alterations associated with peripheral sensory nerve infection are seen as a critical event in the natural history of HD. Because of their inflammatory influence on peripheral nerves, leprosy responses remain a significant contribution to sensory loss and dysfunction once the infection is established.

Genetics of leprosy

Investigation focused on the link between human genetics, the HD genome, and HD susceptibility can help researchers better understand how and why people get the disease. The PARK2/ PACRG gene (placed on chromosome 6q25-q27) and the NRAMP1 gene (found on chromosome 2q35) are both linked to leprosy susceptibility. The chromosome 10p13 locus, the TAP1 and TAP2 (transporter associated with antigen processing) genes (on chromosome 6p21), TNF- (tumour necrosis factor alpha) (on chromosome 6p21), and the VDR (vitamin D receptor) gene (on chromosome 12q12) genes, as well as a variety of innate and adaptive immunity factors, have all been linked to HD susceptibility. There have been an increasing number of publications in recent years that show the innate immune response plays a crucial role in predicting susceptibility to leprosy and its reactional stages. Genetic modulation of the innate immune response has been related to higher vulnerability to leprosy and the development of leprosy responses, as evidenced by various polymorphisms of the *NOD2* gene. Genetic diversity in polymorphisms linked with Toll-like receptors demonstrates a critical role for the innate immune system in a dysregulated inflammatory response during leprosy responses. A specific human polymorphism in Toll-like receptor 1 (T1805G variation), which has been linked to poor mycobacterial intracellular signaling, has been found to protect against type 1 responses [1-3].

On a genomic basis, investigations have also proven a link between HD susceptibility and certain characteristics of individual genetics and immunology. With 706 patients and 1,225 controls, a large-scale GWAS (genome-wide association study) was done. CCDC122 (13q14), C13orf31 (13q14), *NOD2* (16q12), TNFSF15 (9q32), HLA-DR (6p21), and RIPK2 are six genes implicated in the innate immune response that have been linked to HD risk (listed with their corresponding chromosomes) (8q21). That research demonstrated that gene variations in the *NOD2*-mediated signaling system are linked to *Mycobacterium leprae* infection susceptibility. Following research, it was shown that the differential expression of certain of these genes is linked to distinct types of HD. *NOD2* promotes monocyte development into dendritic cells, which is linked to the tuberculoid form. In the case of lepromatous leprosy, the expression of galectin-3 is linked to monocyte development into macrophages [4].

Genome sequencing

Mycobacterium leprae is directly connected to Mycobacterium TB, however its genome has experienced reductive evolution, resulting in a genome of just 3.27 Mb, compared to M. tuberculosis' genome of 4.41 Mb. Because some of the genes in *Mycobacterium leprae* were deleted, it became an obligate intracellular pathogen that could not be cultivated on axenic medium and needed the help of a host to live [5]. This makes it difficult to collect enough bacterial DNA for scientific purposes, such as whole genome sequencing (WGS). Nonetheless, the genome of *Mycobacterium leprae* was published for the first time in

Correspondence to: Jeremy Walton, Department of Medicine, University Paris-Sud, Orsay, France, E-mail: jeremy@walton.ulf.edu

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2001, resulting in the categorization of *Mycobacterium leprae* into four genotypes and 16 subtypes (A–P). *Mycobacterium leprae* genome contains a number of repetitive elements, including RLEP, which has 37 copies and is commonly used in molecular diagnostics to detect the presence of this mycobacterium [6].

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