Mycobacterial Diseases
ISSN: 2161-1068
OPEN ACCESS Freely available online
Perspective

Impact of Genetic Studies in Treating *Mycobacterium tuberculosis* Infection

Abhishek Mishra*

Department of Public Health, Federation University, Ballarat, Australia

DESCRIPTION

In 2016, Tuberculosis (TB) caused by the human pathogen *Mycobacterium tuberculosis*, was the leading cause of death due to a single infectious agent, killing 1.6 million people. Droplet nuclei containing *Mycobacterium tuberculosis* are spread through the air from the lungs of people with active disease to the respiratory tracts of people who are not infected. Mycobacterium tuberculosis infection is a multistage process that begins with the first encounter with the bacterium. As a result, a multistep disease course must be imagined. Following inhalation, the droplet nuclei migrate to the alveoli, where they are phagocytosed by alveolar macrophages and dendritic cells. The bacterium's phagocytosis activates a strong host cellular immune response, triggering a series of events involving cytokines and chemokines. Individuals exposed to the bacterium will not all become infected, and many will remain free of infection depending on the infection pressure. The bacteria will begin to replicate in the intracellular environment and migrate to lymph nodes in the lung via the lymphatic system in infected individuals. Cell-mediated immunity develops and tuberculin reactivity develops within the first 2-8 weeks of infection. Granulomas are formed by activated T-lymphocytes and macrophages to limit the spread and replication of bacteria. The majority of people will remain asymptomatic and contain the bacterium, entering a stage known as Latent Tuberculosis Infection (LTBI). It is estimated that in 2014, 25% of the global population was latently infected with *Mycobacterium tuberculosis*. The immune system can contain the infection at this stage, but if it fails, the infection may progress to active disease. Only 5%-15% of immune-competent LTBI positive people will develop clinical TB.

Genetic studies of tuberculosis susceptibility have been on-going for decades, but have gained in recent years due to improved methodological approaches and technological advances. The majority of studies used traditional approaches used in clinical genetics and genetic epidemiology (linkage and association studies), but these encountered difficulties similar to those encountered in genetic investigations of other complex diseases. Polygenicity, the definition of TB phenotypes, and the collection of appropriately large study cohorts with carefully defined homogenous phenotypes are three on-going challenges. Recently, functional genetic studies such as epigenetics, microRNAs, and transcriptomics have shed light on the genetic basis of tuberculosis susceptibility. The early epidemiological evidence is genetic susceptibility of *Mycobacterium tuberculosis* infection and disease progression, but it will also highlight the contributions of genetic epidemiology and functional genetics, as well as controversies, current research gaps, and future developments.

CONCLUSION

The involvement of a human genetic component in susceptibility to *Mycobacterium tuberculosis* infection and progression to active disease is undeniable. Clinical genetics, genetic epidemiology, population genetics, and functional genetics findings have all contributed to the identification of TB susceptibility genes. The other side of the phenotypic coin is more intriguing: resistance to either initial infection or, after infection, resistance to disease progression. Although the phenomenon is now recognised, the precise genetic variants and mechanisms involved remain unknown. The most specific infection and disease phenotypes, and the resister phenotype are identifying action able to genetic variants in tuberculosis infection and disease.

Correspondence to: Abhishek Mishra, Department of Public Health, Federation University, Ballarat, Australia; E-mail: ab.mish@gmail.com

Received: 19-Oct-2022, Manuscript No. MDTL-22-20606; **Editor assigned:** 21-Oct-2022, PreQC No. MDTL-22-20606 (PQ); **Reviewed:** 04-Nov-2022, QC No. MDTL-22-20606; **Revised:** 11-Nov-2022, Manuscript No. MDTL-22-20606 (R); **Published:** 21-Nov-2022, DOI: 10.35248/2161-1068.22.S4.002

Citation: Mishra A (2022) Impact of Genetic Studies in Treating *Mycobacterium tuberculosis* Infection. Mycobact Dis. S4.002.