Mini Review

BIOMEDICAL Journal of Scientific & Technical Research

ISSN: 2574 -1241

DOI: 10.26717/BJSTR.2021.37.005985

Immunology of Tuberculosis: An Old and Persistent Challenge

Denise S Rodrigues*

The Secretary of Health of The State of São Paulo, Instituto Clemente Ferreira, Sao Paulo, Brazil

***Corresponding author:** Denise S Rodrigues, The Secretary of Health of The State of São Paulo, Instituto Clemente Ferreira, Sao Paulo, Brazil.

ARTICLE INFO

Received: July 13, 2021

Published: July 21, 2021

Citation: Denise S Rodrigues. Immunology of Tuberculosis: An Old and Persistent Challenge. Biomed J Sci & Tech Res 37(2)-2021. BJSTR. MS.ID.005985.

ABSTRACT

Tuberculosis remains a public health problem worldwide, even with the advance in new diagnostic methods, the mechanism of interaction between *M. tuberculosis* and the host's immune system is still not fully elucidated.

Introduction

Tuberculosis is a leading cause of morbidity and mortality in the world. About a quarter of the world's population is estimated to be infected with *Mycobacterium tuberculosis*. Globally, an estimated 10.0 million (range, 8.9-11.0 million) people fell ill with TB and 3 million deaths a year [1]. This disease is caused by *Mycobacterium tuberculosis*, an acid-fast bacillus that is transmitted primarily *via* the respiratory route and is spread when people who are sick with tuberculosis expel into the air mainly during coughing. Infection occurs in the lungs, but the organism can seed any organ *via* hematogenous spread. However, only a small minority of individuals with latent infection with *M. tuberculosis* develop active disease [2]. The clinical manifestations of tuberculosis represent a complex interaction between the causative organism, *Mycobacterium tuberculosis*, and the human host immune response.

Host Response

The lung is typically the port of entry and site of active disease although all other organs can be afflicted. An individual with active pulmonary tuberculosis expels small droplets containing tubercle bacilli, which can be inhaled by another individual. If these small droplets enter the alveolar space, they are engulf by pulmonary dendritic cells and macrophages. Some infected macrophages will remain in the lung tissue while some infected dendritic cells will migrate to the draining lymph nodes. T cells in the draining lymph nodes will be activated and on migration recognize the mycobacterial foci in the lung [2,3]. Entry of mycobacteria into phagocytic cells can occur through binding to multiple receptors, all leading to the delivery of the bacilli into macrophage phagosomes. Alveolar macrophages have been shown to play an essential role in the elimination of bacillus; and is considered the first cell population to interact with the tubercle bacillus. Following phagocytosis and replication of pathogenic mycobacteria within macrophages, the infected cells migrate into tissues where additional immune cells are recruited to form a granuloma; this consists predominantly of T cells and *M. tuberculosis*-infected macrophages [2,4]. Granulomatous lesions form and contain the bacteria, preventing development of active disease. The granuloma subsequently develops central areas of necrosis, resulting in the death of the majority of the bacteria and destruction of the surrounding host tissue.

The surviving bacilli exist in a latent state and can become reactivated to develop active disease. The success of pathogenic mycobacteria is largely attributed to their capacity to avoid destruction within host immune cells, in particular macrophages [4]. Protective anti-mycobacterial immune response involves mainly T lymphocytes activating the macrophages and their microbicidal functions through the release of cytokines. This leads to the formation of granulomas, crucial to the containment of mycobacteria. Macrophages/dendritic cells are found in the

centre of these granulomas, along with mycobacteria surrounded by T lymphocytes which provide the proper activation [3,5]. The interaction between the host and the pathogen occurs on different scales. These range from molecular interactions, including, for example, the recognition of specific molecular patterns on innate immune cells by Toll-like receptors, to interactions between individual cells, which, in turn, can range from the phagocytosis of bacteria by macrophages to the spread of disease through a host population and the emergence of different strains of pathogens in response to different host conditions [6].

Conclusion

Tuberculosis is an ancient disease that afflicts the world, understanding the immunopathogenesis of this disease is important for the development of more effective control measures, and novel treatment strategies.

References

1. (2019) Global tuberculosis report. Geneva: World Health Organization.
2. S H E Kaufmann (2004) New issues in tuberculosis Ann. Rheum Dis 63(Suppl 2): 50-56.
3. Edith NG Houben, Liem Nguyen, Jean Pieter (2006) Interaction of pathogenic mycobacteria with the host immune system. Current Opinion in Microbiology 9(1): 76-85.
4. JoAnn M Tufariello, John Chan, JoAnne L Flynn (2003) Latent tuberculosis: mechanisms of host and bacillus that contribute to persistent infection. Lancet Infect Dis 3(9): 578-590.
5. ED Chan, L Heifets, MD Iseman (2000) Immunologic diagnosis of tuberculosis: a review. Tubercle and Lung Disease 80(3): 131-140.
6. Philana Ling Lin, JoAnne L Flynn (2010) Understanding Latent Tuberculosis: A moving target. J Immunol 185(1): 15-22.

ISSN: 2574-1241

DOI: 10.26717/BJSTR.2021.37.005985

Denise S Rodrigues. Biomed J Sci & Tech Res