Opinion

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Immunopathogenesis and Co-morbidities of Tuberculosis

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Abstract

The etiologic agent of *Tuberculosis* (TB), *Mycobacterium tuberculosis*, continues to pose a serious threat to worldwide public health. Globally, 10.4 million new TB cases were recorded in 2016, and there were around 1.7 million TB-related fatalities. One of the most important aspects of attempts to eliminate TB through the creation of potent vaccines and immunological treatments understands the host response to *M. tuberculosis* infection. An intracellular infection called *M. tuberculosis* spreads by the inhalation of aerosolized droplets carrying germs. The lungs' innate immune cells, particularly macrophages, dendritic cells, monocytes, and neutrophils, constitute the first line of defence against *M. tuberculosis*.

Keywords: *M. tuberculosis*, Public health, Infection, Innate immune cells.

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Introduction

A key component of defence against *M. tuberculosis* is the conversion of phagosomes that contain bacteria into acidified, antibacterial compartments. In this context, it is well known that adaptive immune cells like CD4 and CD8 T cells contribute to the antimycobacterial defence by producing IFN-, which can activate infected myeloid cells and suppress bacterial multiplication. The host is able to support *M. tuberculosis* despite pressure from host immunity. Granulomas, which are initially collections of infected and uninfected myeloid cells bounded by a lymphocytic cuff, are the characteristic lesions of *M. tuberculosis* infection [1].

Intended to stop bacterial spread to extra pulmonary locations, the granuloma can also serve as a place for long-term bacterial survival. It is becoming more and more obvious that a number of innate and adaptive immune cells contribute to the immunological response to *M. tuberculosis* infection. *M. tuberculosis* has evolved a variety of techniques to avoid and subvert immune responses in order to stay within a host. For the creation of effective TB vaccines, a better knowledge of the intricate interactions between *M. tuberculosis* and host immunity is required [2]. The only approved vaccine against tuberculosis is Bacille Calmette-Guérin (BCG), an attenuated strain of the mycobacterium bovis that was created over a century ago.

Uncertainty persists regarding the immunological causes of BCG's subpar effectiveness. The induction of antigen-specific CD4 T cells that produce IFN- is another long-held concept regarding the nature of desired immune responses in an ideal TB vaccine that is currently being updated to reflect the growing understanding of host immunity to *M. tuberculosis* infection gleaned from animal models and human cohort studies. The immunological response to *M. tuberculosis* infection is being studied at ever-higher resolutions thanks to developments in imaging and single-cell technologies, high-throughput methods, and systems-based analysis. Opportunities to use knowledge of the immunology of *M. tuberculosis* infection to improve treatments and TB vaccines are growing as understanding of the host response to *M. tuberculosis* infection advances [3].

After inhaling aerosolized droplets carrying live germs into the lungs, *M. tuberculosis* is transmitted. A number of factors, such as the degree and length of contact with a person who has active tuberculosis (ATB), as well as the immunological capacity of the *M. tuberculosis*-infected person, might affect the success of transmission. *M. tuberculosis* infection manifests as a continuum of diseased/infected states ranging from asymptomatic latent TB infection (LTBI) to ATB illness in a clinical context. The elimination of TB has faced particular difficulties as a result of this intricacy and the amazing variability of lesions within single patient. Co-morbidities that alter immune response can aggravate TB illness or hasten the transition of LTBI patients to ATB. HIV infection is the single biggest risk factor for the development of TB, and in latently infected persons, HIV co-infection increases the probability of acquiring TB from a 5–10% lifetime risk to a 10%

yearly risk. Given that HIV-positive people accounted for more than a fifth of all TB-related fatalities in 2016, this statistic emphasises the importance of HIV co-infection to overall TB mortality.

HIV infection causes progressive CD4 T-cell depletion and dysfunction, which suppresses the immune system and impairs resistance to *M. tuberculosis*. It has been noted that peripheral blood and broncheoalveolar lavage (BAL) samples from HIV-infected people with LTBI specifically depleted *M. tuberculosis*-specific CD4 T cells. Numerous studies suggest that increased expression of the HIV co-receptor CCR5 in CD4 T cells from TB patients may lead to selective depletion [4].

Additionally, HIV infection may affect CD8 T-cells and other immunological compartments that have protective immunity against *M. tuberculosis*. For instance, it has been noted that HIV-infected people exhibit decreased proliferation and degranulation of *M. tuberculosis*-specific CD8 T-cells from LTBI patients compared to HIV-uninfected people. Numerous other disorders or behaviours, such as smoking, poor nutrition, diabetes, helminth infections, cancer, and chronic lung diseases, have also been linked in studies to TB [5]. To completely comprehend the foundation of reported connections with other infections and morbidities, more research will be needed.

Conclusion

The difficulties one face is highlighted by the lack of BCG replacements that are adequate. Understanding the interplay between innate and adaptive immunity is crucial for the logical creation of better vaccines since *M. tuberculosis* is skilled at disrupting it. The situation of TB vaccine development is resurgent today more than ever, giving reason for positive expectation for more effective vaccines and therapies against TB, even in the lack of protective correlates and in the face of dismal preliminary findings for MVA85A.

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