



Immune responses to *Mycobacterium tuberculosis* infection



Worldwide, tuberculosis (TB) remains the most frequent and important infectious disease that is responsible for causing significant morbidity and death. About a fourth of the world's population is infected with *Mycobacterium tuberculosis* (*Mtb*), the etiologic agent of TB. The World Health Organization estimates that eight to ten million new cases of TB occur annually worldwide, and the incidence of TB is currently increasing due to the COVID-19 pandemic. In this context, TB is one of the top three (with malaria and HIV) leading causes of death from a single infectious agent, with approximately 1.6 million deaths attributable to TB annually. In particular, pulmonary TB, the most common form of TB, is a highly contagious and life-threatening infection. *Mtb* infection originates in the lungs. Inhaled droplets containing *Mtb* are primarily engulfed by lung macrophages that lead to the release of inflammatory mediators, which attracts other innate immune cells to the site of infection. However, these innate responses are insufficient to control the progression of the infection into symptomatic TB. In most immuno-competent humans, the onset of adaptive immunity, mediated mainly by CD4 and CD8 T-lymphocytes, results in the formation of granulomas where *Mtb* is contained but not eradicated. This condition, where *Mtb* infection persists within the host at a sub-clinical level, has been termed as latent TB (LTBI). However, individuals with LTBI can reactivate to symptomatic disease upon immune-compromising conditions, such as HIV infection. This comprehensive special issue invites cutting edge research findings and reviews on:

Guest Editor

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- (1) The recent advances in the understanding of the host immune responses against TB
- (2) Role of M1 and M2 macrophages in host immune responses against *Mtb* infection
- (3) Role of B cells in host immune responses against *Mtb* infection
- (4) Role of regulatory T cells in host immune responses against *Mtb* infection
- (5) Pathogenesis in TB
- (6) Novel therapies for TB
- (7) COVID-related disruptions in managing TB

SPECIAL ISSUE