



Cellular and Molecular Mechanisms in Mycobacterial Infection 3.0

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Message from the Guest Editors

Mycobacterium tuberculosis (*M.tb*) caused over 10 million cases of tuberculosis (TB) and 1.3 million deaths in 2022. The current TB vaccine, a live attenuated form of *M. bovis* named *M. bovis* Calmette–Guerin (BCG), provides insufficient protection. In addition, the emergence of multiple and extremely drug-resistant strains of *M.tb* is a growing problem. Novel vaccines and drug therapies are urgently required. The primary host cell of *M.tb*, the macrophage, serves as the first line of defense. Multiple pattern-recognition receptors sense mycobacterial molecular patterns, triggering intracellular signaling cascades and induction of proinflammatory cytokines, chemokines, and antimicrobial molecules. Macrophage-derived cytokines such as tumor necrosis factor, interleukin-12, IL-1 β and IL-18 are critical for host defense against tuberculosis. Adaptive responses, including antigen-specific T cells, are critical in controlling the growth of *M. tb* by producing interferon-gamma, activating macrophages, and orchestrating granuloma formation.





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