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Host immune response in tuberculosis
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The caseating granuloma is recognised as the hallmark of the tissue response to infection by Mycobacterium tuberculosis. It is also clear that the immune response to infection is generally excellent since only about 1% of the 1.7 billion infected people worldwide have active disease. However, in those patients with active infection, there is widespread inflammatory tissue destruction. This talk will focus on the role of the innate immune system in both host defence to and the pathology of tuberculosis. The principal innate inflammatory cells involved in host defence are phagocytic cells of the macrophage lineage including multi-nucleate giant cells but others including stromal cells, neutrophils and NK cells are of key importance. The innate immune response acting through a series of pattern recognition receptors orchestrates the host response by secreting a range of mediators, cytokines and chemokines. Innate immunity targets the pathogen by secretion of molecules such as cathelicidins and defensins. In addition, by upregulating gene expression and secretion of matrix metalloproteinase enzymes, it is critical to the destruction of host tissue. This is both the pre-requisite for the spread of infection and the hallmark of severe infection leading to morbidity and mortality. Innate immune function is influenced by the physiological and metabolic environment. In this talk, the diverse aspects of innate immunity will be discussed and the potential for the novel application of immune modulators to improve patient outcomes will be examined.