

Innate Immunity and Induction of Immune Response

先天性免疫力與免疫反應之引發

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Infection of cells by microorganisms activates the inflammatory response. The initial sensing of infection is mediated by innate pattern recognition receptors (PRRs), which include Toll-like receptors (TLRs), RIG-I-like receptors, NOD-like receptors, and C-type lectin receptors. The intracellular signaling cascades triggered by these PRRs lead to transcriptional expression of inflammatory mediators that coordinate the elimination of pathogens and infected cells. However, aberrant activation of this system leads to immunodeficiency, septic shock, or induction of autoimmunity. TLRs are evolutionarily conserved innate receptors expressed in various immune and non-immune cells of the mammalian host. TLRs play a crucial role in defending against pathogenic microbial infection through the induction of inflammatory cytokines and type I interferons. Furthermore, TLRs also play roles in shaping pathogen-specific humoral and cellular adaptive immune responses. Microbial infection initiates complex interactions between the pathogen and the host. Pathogens express several signature molecules, known as pathogen-associated molecular patterns (PAMPs), which are essential for survival and pathogenicity. PAMPs are sensed by evolutionarily conserved, germline-encoded host sensors known as pathogen recognition receptors (PRRs). Recognition of PAMPs by PRRs rapidly triggers an array of anti-microbial immune responses through the induction of various inflammatory cytokines, chemokines and type I interferons. These responses also initiate the development of pathogen-specific, long-lasting adaptive immunity through B and T lymphocytes. Several families of PRRs, including Toll-like receptors (TLRs), RIG-I-like receptors (RLRs), NOD-like receptors (NLRs), and DNA receptors (cytosolic sensors for DNA), are known to play a crucial role in host defense.