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The Role of Toll-Like Receptors in Recognizing the Microbial Infection

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Description

A wide range of microbes, including, bacteria, viruses, fungi, and protists, stand ready to attack the human body and thrive in the nutrient-rich environment which is provided by the body. Fortunately, the immune responses function as a defense mechanism and counterattack by recognizing and destroying foreign invaders. But what alerts the body to danger? How are foreign organisms detected? The discovery of microbial-sensing proteins called Toll-like receptors has been helping to answer such questions and transform our understanding of the response to infection.

A small number of Toll-like receptors can easily detect a broad range of human pathogens, and a variety of other molecules that indicate tissue damage, by a wide process called pattern recognition. These receptors initiate two arms of the immune response: the innate and adaptive responses which work together to fight against infection in mammals. The innate response provides immediate protection. However, it is relatively nonspecific in the mode of attack on pathogens, which results in damaging the healthy tissue if the innate immune response lasts for long time. On the other hand, the adaptive response generates antibody-secreting B cells and cytotoxic T cells which are specific and efficient at targeting pathogen. Unfortunately, this process may take long time to develop than the innate response.

Because Toll-like receptors function as first responders to danger signals, they're centrally significant in research efforts to combat infectious and disease. New strategies for manipulating immune responses depend upon understanding the cell biology of Toll-like receptors, including their structure, cell localization, signal transduction pathways, and expression patterns.

Mammals have several distinct classes of PRRs including Toll-like receptors (TLRs), RIG-I-like receptors (RLRs), Nod-like receptors (NLRs), AIM2-like receptors (ALRs), C-type lectin receptors (CLRs), and intracellular DNA sensors such as cGAS. Among these, TLRs were the primary to be identified, and are the simplest characterized. The TLR family comprises 10 members (TLR1–TLR10) in human and 12 (TLR1–TLR9, TLR11–TLR13) in mouse.

TLRs localize to the cell surface or to intracellular compartments like the ER, endosome, lysosome, or endolysosome, and that they recognize distinct or overlapping PAMPs like lipid, lipoprotein, protein, and nucleic acid. Each TLR consists of an ectodomain with leucine-rich repeats (LRRs) that mediate PAMPs recognition, a transmembrane domain, and a cytoplasmic Toll/IL-1 receptor (TIR) domain that initiates downstream signaling. The ectodomain displays a horseshoe-like structure, and TLRs interact with their respective PAMPs or DAMPs as a homo- or heterodimer along with a co-receptor or accessory molecule.

In natural viral infections multiple PRRs are likely to be engaged in responding to a specific virus, and this appears to be the case for TLRs. Systemic infection-induced cytokine response was affected significantly in the double knock-outs while only partially affected in either single knock-out. A number of studies have shown that a spread of TLR agonists may have a positive effect in anti-viral immunity. For example, a bacterial ligand of TLR2/6, FSl-1, was shown to induce significant resistance to experimental HSV-2 infection. Ampligen, an analogue of the synthetic TLR3 agonist poly (I: C), has been explored for the treatment of HIV.

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