Toll-like receptors (TLRs) represent a powerful system for the recognition and elimination of pathogen-associated molecular patterns (PAMPs) from bacteria, viruses, and other pathogens and damage-associated molecular patterns (DAMPs) released from dying cells. Typical PAMPs include bacterial cell wall components, viral pathogens, or pathogenic nucleic acids, including viral RNA and DNA. Activation of TLRs leads to the production of proinflammatory cytokines and type I interferons which are important for the induction of the host immune response against bacterial and viral infections. However, dysregulation and overstimulation can be detrimental, leading to hyper-inflammation, sepsis, and loss of tissue integrity. The involvement of TLRs in inflammation and bacterial infection has been recognized for a long time. There is an increasing number of reports demonstrating the involvement of TLR activation in a variety of viral infections, associated with protective immunity, but also immune hyperactivation and even viral replication. Recent data show the involvement of TLR activation in various acute respiratory viral infections, including SARS-CoV-2 and indicate an essential role in COVID-19 pathology. It aimed to gather newest data and hypotheses regarding molecular and cellular mechanisms of TLR triggering and activation and their downstream signaling pathways by viral infections, and their correlation to immunology and pathophysiology of the associated diseases, to facilitate translational research resulting in new targets for the treatment of viral infectious diseases including COVID-19.
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