



Toll like Receptor in Infectious Diseases

Yang Wang*

Department of Medical Microbiology and Immunology, Creighton University, Omaha, USA

*Corresponding author: Yang Wang, Department of Medical Microbiology and Immunology, School of Medicine, Creighton University, Omaha, NE 68178; E-mail: ivywang@creighton.edu

Received date: September 04, 2016; Accepted date: September 05, 2016; Published date: September 12, 2016

Copyright: ©2016 Wang Y, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Editorial Note

Many studies reported that genetic variation in interleukin-1 (IL-1) receptors/Toll like receptors (TLRs) are susceptible to infectious diseases. TLRs play an important role in the innate immune response to invading pathogens. TLRs are emerging in conditions such as asthma, rheumatoid arthritis, sepsis syndrome, and systemic lupus erythematosus, suggesting that targeting of TLRs might be useful therapeutically.

TLRs are functioned by Toll/IL-1 receptor (TIR) domain, which occurs in the cytosolic region of family members. TLRs subdivided into two groups based on homology to either the Type I IL-1 receptor or Drosophila Toll receptor extracellular domain. Type I IL-1 receptor includes the receptor for the important Th1 cytokine IL-18, and T1/ST2, which may have a role in Th2 cell function. Drosophila Toll receptor includes six mammalian TLRs, including TLR2 and TLR4, that largely mediate the host response to gram-positive and gram-negative bacteria, respectively. Signaling pathways activated via the TIR domain trigger the activation of downstream kinases, and transcription factors such as NF- κ B, and involve the adaptor protein MyD88, which contains a TIR domain itself [1]. Most of the inflammatory responses downstream of TLRs are dependent on a common signaling pathway mediated by the adaptor molecule MyD88 [2].

Toll-like receptors (TLRs) recognize common microbial or host-derived macromolecules and have important roles in early activation of the immune system [3]. Many of the pathophysiological processes involved in cardiovascular, autoimmune, and other inflammatory conditions involve TLR activation, and there is increasing evidence for modulation of TLR expression in such diseases. Many TLR isoforms have been implicated in a wide variety of diseases [4]. Therefore, better understanding of the mechanisms of TLR-targeting therapies will allow more specific treatments to be developed for infectious diseases. Ongoing studies are expected to further clarify the role of genetic variation and disease susceptibility in this important class of innate receptors, and provide important clues for therapeutic targeting of TLRs for therapeutic interventions and treatment of infectious and inflammatory diseases [5].

References

1. Bowie A, O'Neill LA (2000) The interleukin-1 receptor/Toll-like receptor superfamily: signal generators for pro-inflammatory interleukins and microbial products. *J Leukoc Biol* 67:508-514.
2. Pandey RK, Yu FS, Kumar A (2013) Targeting toll-like receptor signaling as a novel approach to prevent ocular infectious diseases. *Indian J Med Res* 138:609-619.
3. Maglione PJ, Simchoni N, Cunningham-Rundles C (2015) Toll-like receptor signaling in primary immune deficiencies. *Ann N Y Acad Sci* 1356:1-21.
4. Kay E, Scotland RS, Whiteford JR (2014) Toll-like receptors: Role in inflammation and therapeutic potential. *Biofactors* 40:284-294.
5. Medvedev AE (2013) Toll-like receptor polymorphisms, inflammatory and infectious diseases, allergies, and cancer. *J Interferon Cytokine Res* 33:467-484.