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Editorial

Toll-Like Receptors as a Therapeutic Target From Past to Future

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Introduction

In the context of human and mammalian immunology, Toll-like receptors (TLRs) as an innate immune receptor were first distinguished on human macrophages in 1997. TLR expression was found to be expressed by cells of adaptive immunity such as T cells in addition to innate immune cells in subsequent immunology studies.[1] In addition to having a direct impact on the biology of the innate immune response, B cells and TLRs have been shown to affect the development, activation, survival, and associated immune response of various types of T cells and B cells.[2] TLRs, on the other hand, were found to be expressed by newly discovered cells known as innate lymphoid cells (ILCs), which resemble lymphoid cells but are actually a different form of hematopoietic innate immune cells.[3-5] These ILCs, for instance, express some of the TLRs such as TLR2 and TLR3, but they do not express TLR4 or TLR7.[6-8] In addition, a specific population of ILCs known as c-Kit + which was found to expand in humans during filarial worm or helminthic infection expresses TLR5, TLR7, and TLR9. In response to the TLR6 ligand, these purified c-Kit + ILCs produce a greater amount of granulocyte-macrophage colony-stimulating factor. However, no additional prototypic cytokines such as IL-5, IL-17A, or interferon-γ were produced by TLR-signaling in the c-Kit + ILCs. Nevertheless, cytokine production from human type 3 ILCs is only triggered by TLR2 agonists when IL-2, IL-15, and IL-23 are present.[9-11] In simian immunodeficiency virus-infected macaques, this specific expression of TLR2 on type 3 ILCs or lymphoid tissue inducer cells is responsible for inducing the apoptosis of these cells. Cancer and autoimmunity are two examples of sterile inflammatory conditions in which TLRs play a significant role. For instance, they have been ensnared in the pathogenesis of a few auto-safe sicknesses, including systemic lupus erythematosus, rheumatoid arthritis, seronegative spondyloarthritides, multiple sclerosis, experimental autoimmune encephalomyelitis, type 2 diabetes mellitus, foundational sclerosis, Sjogren's syndrome, and myositis (a persistent provocative immune system illness principally influencing skeletal muscles, causing serious muscle shortcoming and weariness). They have additionally been related to the pathogenesis of a few human diseases, including B cell malignancies, colorectal disease, basal cell carcinoma, bladder malignant growth, and different tumors.[12] New immunotherapies for autoimmune and cancer diseases have been developed as a result of TLRs' involvement in their pathogenesis. To improve the effectiveness of radiation therapy in halting tumor growth, even TLR-mediated immune stimulation is being utilized. For instance, a number of TLRs have recently been linked to the pathogenesis of neurodegenerative conditions as well as the mechanisms by which these conditions can be repaired.[13-15] As a

Author's Biosketch

Professor Xu Huaxi is the Vice president of Jiangsu University. He is a Ph.D supervisor, the pace-setter of the provincial key academic discipline of clinical examination diagnosis who enjoys the state council special allowance. He is also the Director of the Institute of Clinical Laboratory Medicine and Academic leader of the Qinglan Project of Jiangsu province. Moreover, he is the executive member of the Chinese Society of Immunology (the deputy chairman of the Jiangsu Society of Immunology), the executive member of the Chinese Society of Microbiology, the vice chief member of the Jiangsu Society of Microbiology and Immunology of Chinese Medical Association, and a member of the Ministry of Public Health Guiding Committee of College Medical Textbook Construction. In addition, he is the editor-in-chief of the Journal of Jiangsu University (Medicine Edition), a member of the editing board of Shiyong Laonian Yixue and the Chinese Pharmacological Bulletin. He has supervised 18 Doctoral candidates and more than 20 Master's degree candidates.

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result, therapeutic approaches to infectious and non-infectious diseases target TLRs because of their significant role. Even some studies have demonstrated that these TLRs can regulate the processes of steroidogenesis and spermatogenesis in males and ovulation, fertilization, gestation, and parturition in females, respectively, to control reproduction in mammals. Regeneration (e.g., liver regeneration in mammals) has also been linked to TLRs. Acute and chronic rejection of organ transplants is also facilitated by their involvement in allograft inflammation. TLRs have been discovered in humans for 20 years, and their roles in infections, inflammation, reproduction, development, autoimmunity, cancer, allograft inflammation/rejection, and regeneration are now ruling human biology.[16-18] Nevertheless, the unexplored role of TLR in extremely complex mammalian/human biology can be explored through ongoing research in the field of TLRs. TLRs are amazing, but their evolution and the mammals' biology under homeostatic and inflammatory disease conditions need further study.[19] As a result, a variety of TLR agonists and antagonists have been developed to target a variety of inflammatory conditions, including cancer and autoimmunity, in addition to controlling various biological functions.[20]

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