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New insights into Patho-mechanisms of Toll-Like Receptors in Autoimmune Diseases

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ABSTRACT

Toll-like receptors (TLRs) are supposed to be crucial in the pathogenesis of autoimmune diseases, according to several studies. The pattern recognition receptors (PRRs) family that includes the TLRs is capable of identifying a variety of pathogen-associated molecular patterns (PAMPs), but they can also identify endogenous molecules. TLRs are found on various cellular membranes and are type I transmembrane proteins. The intracellular group is found in cellular compartments that are responsible for recognizing nucleic acids, whereas the extracellular group is found on the plasma membrane. They're generated by the inflammatory cells and trigger numerous intracellular pathways, which leads to the creation of pro-inflammatory cytokines and chemokines and the expression of co-stimulatory molecules to defend against inflammatory microorganisms. The finding and subsequent characterization of endogenous ligands for TLRs (and other PRRs) provides a unique approach for exploring the origins of autoimmune illness. The review addresses the relationship between TLR activation and subsequent events in the genesis or progression of systemic autoimmune disorders. TLR inhibitors could be a potential technique for treating autoimmune disorders.

Key words: Immune response, autoimmune disease, Toll-like receptor, Therapeutic application.

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INTRODUCTION

In the case when the immune responses to the host are improperly directed, autoimmune diseases result [1]. Systemic diseases or organ-nonspecific (determined by responses to the antigens found in various organs.) and organ-specific diseases (where the T cells and antibodies react to self-antigens in specific tissues) are among such diseases [2]. Abnormal immune system processes are implicated in disease onset and perpetuation, based on the mechanism of immune responses gone awry in the pathophysiology of autoimmune diseases [3]. According to latest epidemiological studies, there are few treatment options for autoimmune diseases, which afflict roughly 5-8% of the world's population. There are around 80 known autoimmune diseases, which are regarded as a significant global socioeconomic problem since they significantly impair patients' quality of life and give them great suffering [1,2]. The transmembrane receptor family known as TLRs is essential for both adaptive and innate immune responses. Besides identifying particular molecular patterns linked to various pathogens, TLRs could identify a variety of endogenous nucleic acids as well as self-proteins. Activating TLRs causes an increase in expressing many inflammatory genes, which fight infection. Inappropriate activation of pathways of TLR by endogenous or exogenous ligands might initiate and/or perpetuate auto immune reactions and tissue damage, based on data that had been derived primarily from human patients and animal models of autoimmune illness [4]. TLRs as well as their signaling pathways become attractive targets for therapeutics because of their significant role in infectious as well as non-infectious disease processes. In our review, we illustrate the potential involvement of TLR pathways in autoimmune disorders and their potential therapeutic use.

TLRs: History, Structure, and Functions

The functional characterization and identification regarding the toll gene and its protein in Drosophila marked the beginning of the discovery of TLR receptors [5]. TLRs have been named due to their resemblance to toll protein in their function as receptors of the cell surfaces [6]. These receptors are involved in dorsoventral polarization, embryonic development [7], and immunity against gram-positive fungal and bacterial infections [8]. Type I integral transmembrane proteins, which the TLRs are a subtype of, usually have 3 domains, which are a

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C-terminal domain (CTD) facing the cytoplasm, a central single helix transmembrane domain that crosses the membrane, and an N-terminal domain (NTD) that lies outside the membrane (Figure 1) [9,10]. While CTD interacts with several adaptors of signal transduction in order to initiate downstream signaling via its toll-IL1 receptor (TIR) homologous domain, the N-terminal domain forms an ectodomain that functions as a site of ligand recognition for the variety of PAMP [11, 12]. Approximately 19–25 highly conserved short tandem LRR (i.e., leucine-rich repeat) motifs make up the ectodomain's folded solenoid structure, resembling a horseshoe, and give TLRs the much-needed recognizability and specificity for the PAMPs/DAMPs [13]. The 24-29 amino acids that make up each TLR LRR repeat sequence are arranged in the pattern xLxxLxLxx [14]. Furthermore, glycan moieties found in the NTD operate as real binding sites for a variety of ligands that are derived from pathogens [15].

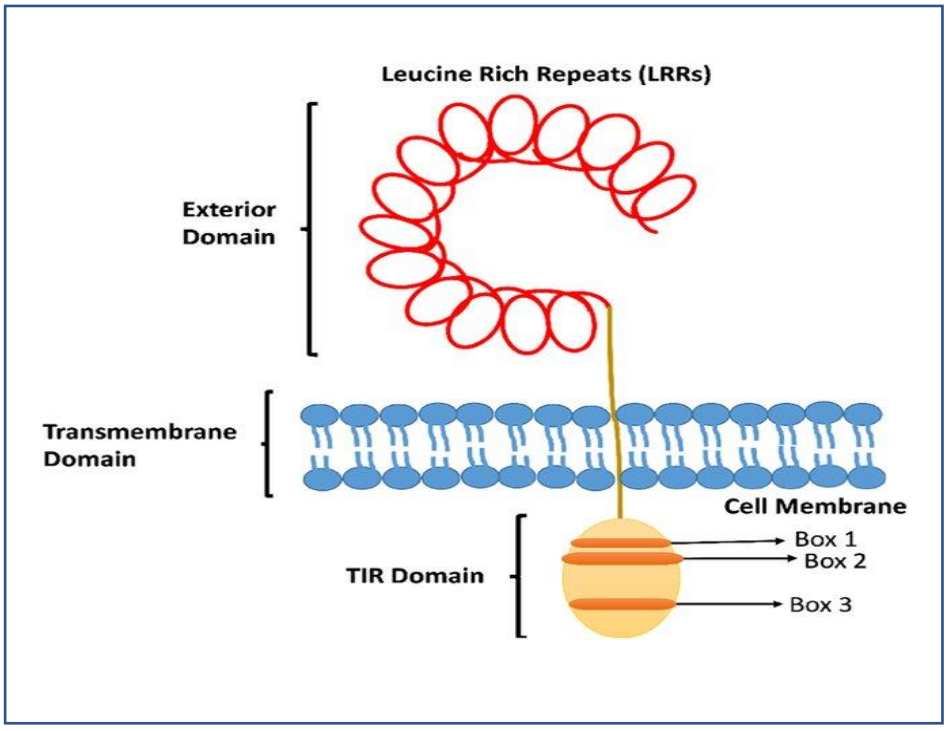

Figure 1. Basic structure of TLRS [9].

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TLR Signaling Pathway

TLR signaling pathway consists of 2 main path-ways, which are: MyD88-dependent path-way and TRIF-dependent path-way (figure 2), both of which play critical roles in immune responses [16, 17].

MyD88-Dependent Pathway

Apart from TLR3, the majority of TLRs use such pathway. TLRs recruit the adaptor protein MyD88 upon ligand binding for initiating a signaling cascade. MyD88 associates with interleukin-1 receptor-associated kinases (IRAKs), therefore be the forerunner to the activation of the tumor necrosis factor receptor-associated factor 6 (TRAF-6) [18]. IκB is phosphorylated and degraded in such cascade for releasing nuclear factor kappa-light-chain-enhancer of stimulated B cells (NF-κB), which translocation to nucleus releases. IL-6 and TNF- are among the pro-inflammatory cytokines produced by this pathway [19].

TRIF-Dependent Pathway

TLR-3 and, under some conditions, TLR-4 mostly use this pathway. These TLRs first recruit the adaptor protein TRIF, which activates TANK-binding kinase 1 (TBK-1) and receptor-interacting protein 1 (RIP-1). Interferon regulatory factor 3 (IRF-3) phosphoryzes in response, after that trans locates to the nucleus. The TRIF-dependent pathway promotes type I interferon's production, which are highly important for the anti-viral responses [20].

Key Components in TLR Signaling

- Adaptor Proteins: MyD88, TRIF, TIRAP (also known as Mal), and TRAM play critical roles in recruiting and activating downstream signaling molecules.
- Kinases: IRAKs, TBK1, and IKK complexes are pivotal in propagating signals from the receptor to the nucleus.
- Transcription Factors: NF-κB and IRFs (e.g., IRF3) regulate transcription of genes that are involved in inflammatory as well as antiviral responses [21].

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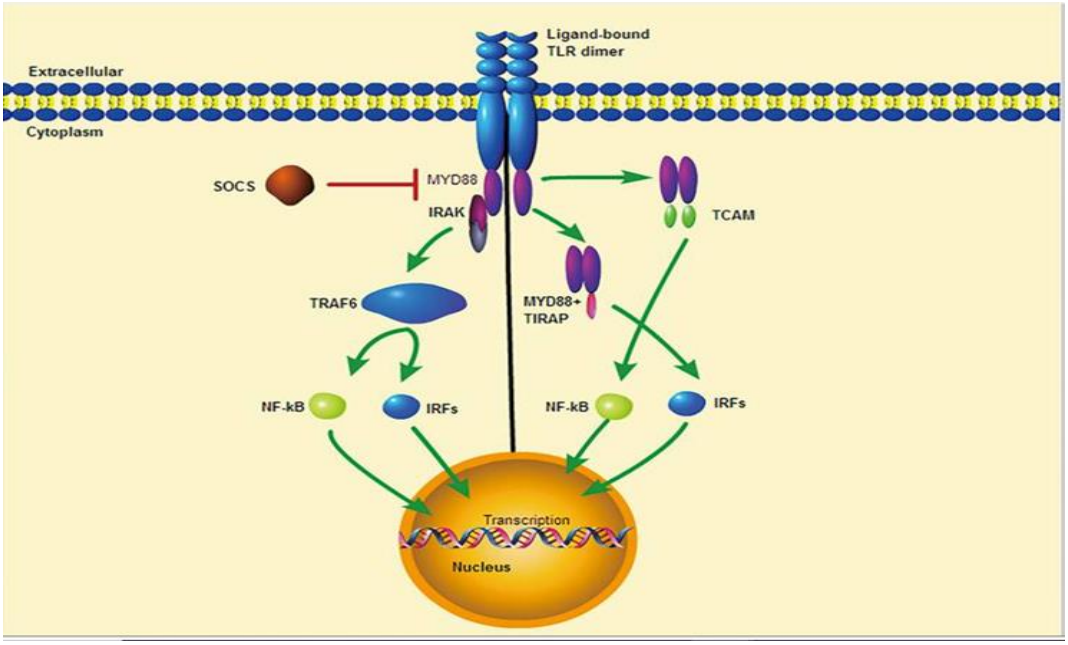

Figure 2. The signaling path. MyD88-dependent path-way (left) and TRIF-dependent path-way (right) [17].

Significance of TLR Signaling Pathways

TLR signaling is critical for host defense as it detects microbial infections and activates proper immune responses. However, deregulation of these pathways can cause excessive inflammation, which contributes to disorders like sepsis and chronic inflammatory diseases. Modulating TLR pathways has significant therapeutic options for treating infectious diseases, autoimmune disorders, and cancer [22, 23].

TLRs in Autoimmune Disease Pathogenesis

Numerous coincidental mechanisms related to the presence of auto-reactive immune cell subsets and lack of immunological tolerance give rise to autoimmune disorders [24]. Through the up-regulation of antigen-presenting cell (APC) co-stimulatory molecules and the induction of pro-inflammatory cytokine production, TLR signaling is crucial for the activation of the adaptive immune system [25].

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A number of autoimmune illnesses can be induced by stimulating Toll-like receptors (TLRs), a group of pattern recognition receptors (PRRs) that include damage-associated molecular patterns (DAMPs) and pathogen-associated molecular patterns (PAMPs). However, in some animal models, activation of particular TLRs can both cause and prevent autoimmune disorders. Both hematopoietic and non-hematopoietic cells exhibit combinations of distinct TLRs, which contribute to the induction of autoimmunity or tolerance. Activating or inhibiting responses is determined by the coordination of signals from several TLRs, other PRRs, and membrane-standing receptors both inside and between cells [26, 27].

Clinical Trials and Emerging Therapies

Choosing the appropriate drug is crucial to the long-term result for patients with systemic autoimmune disorders, which necessitate lifetime therapy. Despite the availability of several treatments, a considerable percentage of patients do not experience the intended full remission. However, because so many medications have been successfully tested in clinical trials, the variety of accessible therapy is always growing.

TLR 7 or 8 inhibitors or both together, TLR antagonists and their signaling pathways are still research target for the treatment of autoimmune diseases. Synthetic oligonucleotides were used in the development of TLR antagonists, including TLR7 and TLR9 inhibitors, TLR7, TLR8, and TLR9. However, these inhibitors are given as subcutaneous injections and require repeated injections which are not feasible for clinical application [24,25]. Its early results have been released, but its potential remains unknown. [26] Although they have not yet undergone in vivo testing, small compounds that exclusively target TLR7 or TLR8 have already been informed[27, 28]. Table 1 shows some of the drugs used to treat a number of autoimmune diseases.

Table 1. Examples of some drugs used to treat a number of autoimmune diseases.

NO	Drugs	Target(TLRs)	Diseases	Year	Ref.
1	DV-1079	7/8/9	Multiple sclerosis	2005	28
2	Hydroxychloroquine	7/9	Autoimmune Diseases, Sjogren's Syndrome, Dry Eye/dry mouth of Primary	2006	29
3	IMO-3100	7/8	Systemic Lupus Erythematosus	2009	30
4	IRS-954	7/9	Rheumatoid Arthritis	2010	31
5	IMO-8400	7/8/9	Plaque psoriasis	2017	32

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While TLR7 responses are marked by a large production of type I IFNs, including IFNa, which seems to play a significant role in autoimmune illnesses, TLR8 induces incredibly low amounts of IFNa but creates a robust production of other pro-inflammatory cytokines [33, 34, 35].

Conclusions

TLRs are crucial for the immune system's ability to detect and respond to infections, and dysregulation of these receptors can lead to the onset and progression of autoimmune diseases. Controlling TLR signals opens new horizons in developing treatments that regulate the immune response to certain autoimmune diseases such as type 1 diabetes, multiple sclerosis, rheumatoid arthritis, and lupus. It is preferable to use combination therapy instead of therapy that targets only one goal in order to avoid the overlapping pathways of some autoimmune diseases. Although TLR-based therapeutics discovery seems challenging, it is exciting and will create new opportunities for drug discovery, development, and prevention of infectious diseases, cancer, allergies, asthma, and autoimmune diseases, either directly or through the creation of better vaccines.

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