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Research Progress on the Role of IL-17 in Spinal Cord Injury

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Abstract: *Spinal cord injury (SCI) is a severe central nervous system injury that often causes permanent motor and sensory function loss. In recent years, increasing studies have focused on the role of the immune system in SCI, especially that of interleukin-17 (IL-17), a key cytokine. Previous research has shown that IL-17 plays important roles in the inflammatory response and tissue repair following SCI, suggesting its potential as a therapeutic target. This review aims to explore the research progress regarding the role of IL-17 in spinal cord injury and its potential therapeutic applications.*

Keywords: *Spinal Cord Injury; Immunity; Interleukin-17*

1. Introduction to Interleukin-17

1.1 The IL-17 Family

The IL-17 cytokine family comprises six structurally related cytokines, designated IL-17A through IL-17F. Among these, IL-17A (commonly referred to as IL-17) has attracted extensive attention due to its pro-inflammatory role in autoimmune diseases. As the first identified member of the IL-17 family [1, 2], IL-17A is a disulfide-linked dimeric glycoprotein composed of 155 amino acids with a molecular mass of approximately 35 kDa. IL-17A is mainly produced by activated memory $CD4^+$ T lymphocytes, but can also be secreted by $CD8^+$ memory T lymphocytes. It exerts strong pro-inflammatory effects and acts as a fine-tuning regulator of the inflammatory response. [3]. Its primary functions include: Promoting local production of chemokines such as IL-8, monocyte chemoattractant protein-1, and growth-regulated proteins[4].Stimulating the production of IL-6 and prostaglandin E2, thereby amplifying local inflammation [5]. Inducing cell-surface intercellular adhesion molecules and driving T-cell responses. It promotes the proliferation and differentiation of neutrophils, the differentiation and maturation of dendritic cell precursor cells, and increases the expression of CD11c, CD40, CD80, CD86, and MHC class II antigens in cells. It is an important component of the connection between the hematopoietic system and the immune system. [6-8]

Other IL-17 family members (IL-17B to IL-17F) and IL-17A are dimers with a conserved amino acid sequence in the C-terminal region. IL-17F exhibits the highest homology with IL-17A, followed by IL-17B, IL-17D, IL-17C, and IL-17E [9, 10]. IL-17A and IL-17F are regulated by modulating the action of IL-23, contributing in part to neutrophil activation by inducing CX chemical factors and granule production and increasing local survival. Furthermore, IL-17A and IL-17F participate in regulating inflammation and host defense. IL-17A and IL-17 family members are associated with inflammation, and they play an important role in the body's immune response and inflammatory response. [11]

1.2 Sources of IL-17

The inflammatory function of IL-17 was initially characterized in mouse models of autoimmune diseases, with an initial focus on IL-17-secreting $CD4^+$ T cells, namely T helper 17 cells (Th7), as key producers of this cytokine. It has been found that $CD8^+$T cells, γδT cells, innate lymphoid cells (ILCs), natural killer (NK) cells, iNK T cells, mast cells and Paneth cells can also be the source of IL-17. $CD4^+$ T helper 17(TH17) cells are the major source of IL-17A and can also produce IL-17F, granulocyte-macrophage colony stimulating factor (GM-CSF), IL-21, IL-22, IFNγ, and tumor necrosis factor (TNF)

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[12, 13]. These mediators act synergistically to exert pro-inflammatory effects. IL-6 and TGF-β were originally identified as key factors driving Th17 cell differentiation.[14-16]. Later studies confirmed that IL-23 in combination with IL-1 or IL-18, together with T-cell receptor (TCR) ligation, promotes Th17 cell activation in both mice and humans.[17-19]. The population of CD4+ T cells that produce IL-17 in the absence of TCR binding is referred to as native TH17 cells. These cells differentiate in the thymus, express the transcription factors RORγt, IL-23R, α4β1 integrin, and CCR6, produce IL-17A, IFNγ, and IL-22, and develop in the absence of IL-6 required for inducible Th17 cells. Natural TH17 cells mediate host protection at mucosal surfaces by producing IL-17 and IL-22[20, 21]. Il-17-secreting CD8+ T cells produce a similar range of cytokines and activate TH17 cells in a similar manner. Il-17-secreting γδT cells produce the same range of cytokines as TH17 cells but are activated by IL-1β and IL-23 in the absence of TCR stimulation[22, 23]. "A new population of T cells that coexpress αβ and γδTCRs and high levels of IL-1 and IL-23 receptors produce IL-17A, GM-CSF, and IFNγ in the presence or absence of TCR-stimulated Il-1β and IL-23 stimulation [24]".Although T-cell receptor activation is critical for IL-17 production by CD4+ and CD8+T cells, production of IL-17 by innate immune cells is primarily driven by inflammatory cytokines, particularly IL-1β and IL-23. In addition, it has also been suggested that neutrophils may also be a source of IL-17 during infection.

1.3 IL-17-related signaling pathways

IL-17 acts mainly by binding to IL-17 receptor and activating downstream signaling pathways.

(1) NF-κB signaling pathway: IL-17 can promote the epithelial-mesenchymal transition of tumor cells, and promote the invasion and metastasis of tumor cells through NF-κB. IL-17 stimulation can increase the phosphorylation levels of Akt1 and NF-κB-p65 in glioma cells. Knockdown or inhibition of PI3K, Akt1 and NF-κB-p65 can also reduce the proliferation and migration of glioma cells induced by IL-17. In addition, PI3K/Akt1 is an upstream regulator of NF-κB-p65 activation in glioma cells incubated with IL-17. Inhibition of PI3K, Akt1, and NF-κB-p65 significantly inhibited IL-17-induced tumor formation in glioma cells. This suggests that IL-17 can promote proliferation and migration of glioma cells through PI3K/Akt1/NF-κB-p65 activation [25]

(2) MAPK signaling pathway: In animal experiments, hyperoxia-induced acute lung injury can be attenuated by regulating the Act1-TRAF6-p38 MAPK pathway through IL-17A-mediated ferroptosis of alveolar epithelial cells [26]. In addition, MAPK can promote the proliferation of vascular endothelial cells and angiogenesis. PD98059, a specific inhibitor of MEK1/2, completely inhibited IL-17-induced ERK2 phosphorylation and significantly inhibited ERK1 phosphorylation.

(3) ERK signaling pathway: in clinical studies, ERK1/2 phosphorylation occurred after IL-17 stimulation in A549 lung cancer cells [27]. IL-17 also promoted lymphangiogenesis by up-regulating the expression of vascular endothelial growth factor (VEGF) -C through the ERK1/2 pathway

(4) STAT signaling pathway: STAT3, as a very important member of the STAT family, is involved in the regulation of cell proliferation, apoptosis and differentiation. Under the conditions of rheumatoid arthritis, various cytokines (cytokines such as TNF-α, IL-1, IL-6 and IL-17 and HIF-1α) activate JAK/STAT3 through IL-6 and affect the maturation and differentiation of osteoblasts. After activation of JAK/STAT pathway, the expression of NF-κB ligand receptor activator (RANKL) was upregulated, and the expression of OPG was inhibited. After RANKL binds RANK on the surface of osteoclast precursors, the NF-κB signaling pathway is activated and promotes osteoclast differentiation. Therefore, cytokines such as IL-17 can lead to bone destruction through STAT signaling [28, 29].

2. The role of IL-17 in SCI

Spinal cord injury is a severe neurological dysfunction that can lead to neuronal degeneration and permanent paralysis. Injury-induced inflammation in turn promotes spinal cord injury. IL-17 plays an important role as an inflammatory factor. It has been shown that motor recovery and tissue retention after spinal cord contusion in mice are significantly improved in the absence of interleukin-17. [30]

2.1 IL-17 exerts its effects by mediating astrocytes

Astrocytes have an important physiological role in central nervous system homeostasis and serve as a bridge between the central nervous system and the immune system. IL-17 and IL-6 are important in many central nervous system diseases characterized by neuroinflammation. However, IL-17 can

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significantly enhance the IL-6 signaling cascade in astrocytes[31].Astrocytes, in response to IL-6, sIL-6R, and IL-17, may shift the production of chemokines to the production favoring the recruitment of T cells to the CNS [32].

IL-17 mediates astrocyte related signaling pathways.IL-17 can induce the upregulation of reactive astrocytes and vascular endothelial growth factor (VEGF) through JAK/STAT signaling, a ubiquitously expressed intracellular signal transduction pathway that is involved in many key biological processes, including cell proliferation, differentiation, apoptosis, and immune regulation. (The role of JAK/STAT signaling pathway and its inhibitors in diseases), and VEGF plays an important role in the repair of spinal cord injury. In the rat experiment, VEGF transfected BMSCS can improve the motor and sensory function of rats with spinal cord injury. (Vascular Endothelial Growth Factor-Transfected Bone Marrow Mesenchymal Stem Cells Improve the Recovery of Motor and Sensory Functions of Rats With Spinal Cord Injury) upregulation of VEGF was induced by IL-17 human astrocytoma cells, IL-17 induces VEGF expression in SCI in vitro and in vivo by activating JAK/STAT signaling pathway. In addition, IL-17 significantly altered tissue preservation and residual urine volume in vivo as well as the integrity of the blood-spinal cord barrier. This newly identified IL-17-JAK/STAT-VEGF axis improves our understanding of the molecular mechanisms of SCI during inflammatory responses and provides another potential target for SCI. IL-17 induces reactive astrocytes and up-regulation of vascular endothelial growth factor (VEGF) through JAK/STAT signaling.) [33] In addition, IL-17 induces MIP-1α expression in primary mouse astrocytes through TRPC channels, IL-17-mediated MIP-1α is induced by binding to cognate IL-17RA, and MIP-1α is involved in astrocyte activation. The role of TRPC channels as novel targets for regulating IL-17-mediated MIP-1α expression and cell activation and has important implications for therapeutic interventions to reverse IL-17-induced neuroinflammation.

2.2 Effect of microRNA in IL-17-mediated spinal cord injury

The aberrant expression of microRNAs after SCI becomes a potential research point. Molecular mechanism of MiR-136-5p targeting NF-κB/A20 in IL-17-mediated inflammatory response after spinal cord injury. In vitro experiments showed that miR-136-5p up-regulated the expression of p-NF-κB by down-regulating the expression of A20, which caused astrocytes to produce inflammatory factors and chemokines. In vivo experiments showed that overexpression of miR-136-5p promoted the production of inflammatory factors, chemokines and p-NF-κB in SCI rats, while inhibiting the expression of A20 protein increased inflammatory cell infiltration and injury in the spinal cord. Therefore, silencing miR-136-5p effectively reduced inflammatory factors and chemokines through the NF-κB/A20 signaling pathway and protected the spinal cord. In contrast, overexpression of miR-136-5p had the opposite effect. In addition, another MicroRNA-155, which is involved in innate and adaptive immune responses, loss of Mir-155 resulted in a significant reduction in the production of IL-17 by T cells. [34] In addition, the suppression of Th17 differentiation induced by Mir-155 deficiency was partially dependent on the increased expression of SOCS1. It has been shown that in SCI, Mir-155 deficiency significantly inhibits Th17 cell differentiation and improves motor recovery after SCI. [35]

2.3 IL-17 promotes spinal cord neuroinflammation after spinal cord injury in rats by activating transcription factor STAT3

After SCI, IL-17 and p-STAT3 levels were significantly increased in spinal cord tissue, peaking at 24 hours. Studies have shown that IL-17 activation via STAT3 contributes to SCI-induced inflammation. Serum analysis showed increased levels of cytokines such as IL-6, IL-21, and IL-23 levels after SCI, supporting a role for IL-17 in promoting an inflammatory environment. As assessed by BBB score and histopathological examination, changes in IL-17, STAT3 activation and related cytokine levels were correlated with the severity of SCI. Studies have found that IL-17 plays a crucial role in promoting neuroinflammation after SCI by activating STAT3. This activation leads to increased expression of inflammatory cytokines, exacerbating the effects of injury. These findings suggest that it is possible to reduce inflammatory responses, limit secondary injury, and promote recovery after SCI by targeting the IL-17 or STAT3 pathway, and that targeting the IL-17/STAT3 pathway could provide a therapeutic approach to alleviate inflammation and potentially improve outcomes after SCI.[36]

2.4 IL-17A can regulate the proliferation and functional recovery of ependymal cells and inhibit the proliferation of neural stem cells and the differentiation of neural cells after spinal cord injury in mice

Ependymal cells are considered to have the function of neural stem cells and play a beneficial role

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after spinal cord injury (SCI). Local injection of IL-17A neutralizing antibody in a mouse model of contusion spinal cord injury showed that IL-17A neutralization promoted ependymal cell proliferation, functional recovery and axonal recombination of corticospinal tract and posterior spinal tract. To test whether ependymal cell-specific IL-17A signaling is sufficient to influence the outcome of SCI, one study created ependymal cell-specific strip-shaped il-17ra knockout mice and analyzed their anatomical and functional responses to SCI. Thus, conditional knockdown of IL-17RA in ependymal cells promoted axon growth and functional recovery while increasing mRNA expression of neurotrophic factors. Ependymal cells may contribute to the regenerative process in part by secreting neurotrophic factors, and IL-17A stimulation can negatively modulate this beneficial effect. Molecular modulation of ependymal cells may be a viable strategy to improve functional recovery. [37] It has also been shown that IL-17 has an inhibitory effect on the proliferation of neural stem cells and neural cell differentiation. IL-17 blocks the proliferation of NSC and leads to a significant reduction in the number of astrocytes and OPCs. Thus, in addition to its proinflammatory role in the immune system, IL-17 may also play a direct role in blocking remyelination and nerve repair in the CNS.[38]

2.5 To find new targets and strategies for combined intervention targeting IL-17

Therapeutic strategies to target IL-17 have emerged clinically. Hyperbaric oxygen (HBO) therapy has been confirmed to inhibit the proliferation and activation of IL-17+γδT cells and reduce the release of IL-17 by down-regulating STAT3/RORγt pathway, while increasing the number of ChAT-positive motor neurons, and significantly improving the motor function of the hind limbs of SCI mice. This study for the first time identified IL-17+γδT cells as the core target of HBO in the treatment of SCI, providing theoretical support for clinical non-drug intervention. [39] In addition, local injection of IL-17A neutralizing antibody can block the IL-17/IL-17RA axis signal, relieve the inhibitory effect on ependymal cells, promote their proliferation and secretion of neurotrophic factors, promote the axonal reorganization of the corticospinal tract and the suture spinal tract, and achieve the functional recovery of SCI mice. JAK inhibitors, which target to inhibit the differentiation of Th17 cells, can also alleviate the chronic inflammation of SCI by reducing the secretion of IL-17, providing a new direction for drug intervention.[30, 37]

Biological agents targeting IL-17A, such as secukinumab, have achieved clinical application in autoimmune diseases. They accurately block proinflammatory signals by neutralizing IL-17A, and have the advantages of rapid onset and strong targeting. Based on the results of SCI animal models, such biological agents are speculated to reduce neuroinflammation after SCI through local or systemic administration, and their clinical safety has been verified, which lays the foundation for subsequent SCI clinical transformation research. At the same time, IL-17+γδT cells and STAT3/RORγt pathway as new intervention targets provide a direction for the development of specific immune modulators for SCI, and the research and development of related targeted drugs has become a research hotspot in the field.[36]

3. Outlook

IL-17 is an important proinflammatory cytokine produced by Th17 cells and has been shown to play a key role in a variety of autoimmune diseases and inflammatory responses. Future studies are needed to further reveal the precise molecular mechanism of IL-17 in SCI, especially how it affects VEGF expression through the JAK/STAT signaling pathway and its specific effects on angiogenesis, inflammation and nerve regeneration. In addition, it is necessary to explore the effect of the interaction between IL-17 and other cytokines (such as IL-6 and TNF-α) on SCI repair. Given that IL-17 may have both proinflammatory and proreparative roles, future research could also aim to distinguish the conditions and mechanisms underlying these two effects. Understanding the circumstances and how to regulate the activity of IL-17 to promote its pro-repair effect and inhibit its pro-inflammatory effect, and developing therapeutic strategies targeting IL-17 based on the role of IL-17 in SCI may provide a new direction for the treatment of SCI. This may include the use of IL-17 antibodies or small molecule inhibitors to inhibit its proinflammatory effects, or the development of methods to enhance its proreparative effects. At the same time, the application of other treatment methods, such as stem cell therapy and nerve growth factor, can further improve the treatment effect.

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