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Poster Presentation

The Role of T Helper 17 in Pathogenesis of Multiple Sclerosis

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Abstract

Multiple sclerosis (MS) is an autoimmune disease of the central nervous system (CNS) which causes demyelination of the nerve fibers. The etiology of this disease is not well understood but it is believed that T helpers play a central role in MS. Numerous findings support the view that Th17 cells play an essential role in pathogenesis of MS and IL-17 secreting T (Th17) cells have a role in inflammation and demyelination of the CNS.In some studies suggest that "There was no significant relationship between the serum levels of these cytokines and Expanded Standard Disability Stated Scale (EDSS) and disease Progression Index (PI)". New drugs targeting specific points of the Th17 pathways are already being tested in clinical trials and provide basis for the development of biomarkers to monitor disease activity. Some examples of the results of other studies are given below: 1. TGF-β negatively regulates the differentiation of encephalitogenic Th17 cells. 2. miR-27a may probably inhibit negative regulators of Th17 cell differentiation, thus promoting its differentiation while miR-214 has an adverse effect. Also both miR-141 and miR-200a show up-regulation in relapsing phase of MS patients compared to remitting and control groups. 3. IFN-β inhibits the expansion of Th17 cells in active multiple sclerosis. 4. JAK2 as a critical factor that stabilizes IFN-γR2 surface expression in Th17 cells from AMS patients, making them sensitive to IFN-γ. 5. Vitamin A modulate the imbalance of Th17 and Treg cells through multiple molecular pathways. 6. IL-11 as a new Th17-promoting cytokine. 7. CXCR3 signaling in glial cells in negatively regulating Th17 cell expansion during EAE. According to these results focusing on the role of Th17 cells and use of its pathways and their biomarkers of diagnosis and disease activity are new windows to effective therapies.

Keywords: Multiple sclerosis, Pathogenesis, Th17

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