

Significant Association between Myelin Proteolipid Protein (58-74) Putative Epitope and Multiple Sclerosis Pathogenesis

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Background: Multiple sclerosis (MS) is the most common autoimmune disease of the central nervous system (CNS). The main cause of MS hasn't revealed yet, but the most probable theory is according to the molecular mimicry that concluded some infections can activate T cells against brain auto-antigens and initiate the disease cascade. Myelin proteolipid protein (PLP) is one of the brain auto-antigens and its restriction and abundance in CNS, turns it to a proper candidate as an auto-antigen. This study is conducted to evaluate of PLP58-72 activatory effects on T lymphocytes and humeral immunity.

Materials & methods: PLP58-72 was predicted as an immunodominant epitope by Meta prediction method using bioinformatics tools. Patients' and healthy individuals' peripheral blood mononuclear cells (PBMCs) were treated by this epitope and its proliferative effects were evaluated through proliferating cell nuclear antigen (PCNA) gene expression changes assessment by real time PCR and immunocytochemistry assay. Finally, the rate of CD4+ and CD8+ T cells was assessed by flowcytometry; ELISA was also performed to measure anti PLP58-72 antibody in patients' serum.

Results: PLP58-72 induced proliferation in patients PBMCs; whereas, peptide didn't influence healthy individuals' PBMCs. CD4+ T cells was the

main activated cells in reaction to PLP58-72 and they arise from 22% to 39.91 %. In addition, immune assay showed 19 fold increase in specific anti PLP58-72 IgG in patients than healthy controls.

Conclusions: It seems that PLP58-72 can stimulate CD4+ T cells and humeral immunityreaction exist in MS patients. Moreover, based on the obtained results, performing such studies can be promising in realizing of MS incidence reasons. As it showed that some microorganisms' epitopes mimicked PLP58-72 may have a potential role in initiation of MS.

Key words: Multiple Sclerosis, Myelin Proteolipid Protein, Molecular Mimicry, Experimental Autoimmune Encephalomyelitis, auto-immune disease