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TIM-3 gene polymorphisms and risk of multiple sclerosis

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Multiple sclerosis (MS) is an autoimmune disease of the central nervous system (CNS). It would be caused by auto reactive T-cells directed against myelin antigens. T-cell immunoglobulin mucin-3 (TIM-3) is a negative regulator glycoprotein expressed by a range of immune cells, including, Th1 cells, activated CD8+ T-cells and in a lower level on Th17 cells. TIM-3 might have a key role in the autoimmune diseases by interacting with TIM-3 ligand to regulate T-cells responses. A defect in TIM-3 regulation has been shown in multiple sclerosis patients. In humans, several single nucleotide polymorphisms (SNPs) have been identified in the TIM-3 gene and are associated with inflammatory diseases. The present study analyzes the association between TIM-3 -574A>C and -1516 C>A gene polymorphisms and susceptibility to MS. DNA samples from 102 patients and 102 healthy controls were genotyped using RFLP-PCR method. Analysis of the alleles and genotypes revealed a significant higher frequency of C/C and lower frequency of A/C genotypes for -574 locus of TIM-3 gene in MS patients (P=0.0002). We also found that C/C genotype for locus of -1516 increased in MS patients, while A/C genotype decreased (P=0.012). Allele C of -574C/C and -1516 C>A SNPs were also more frequent in MS patients (P=0.036 and 0.0027 respectively). These findings suggest that -574 A>C and -1516 C>A polymorphisms in the promoter region of TIM3 gene may affect the disease susceptibility.

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Investigation of intracellular tau modifications and cell-based sensors for studying tau aggregation

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Dysregulation or mutation of miRNAs has been linked to autoimmune diseases, such as multiple sclerosis and experimental autoimmune encephalomyelitis (EAE). However, the meaning of the alteration in miRNA expression level remains unclear. Our purpose was to determine the pattern of miRNA-193a expression throughout relapse and remission phases of EAE. In this study, we induced EAE by immunizing C57BL/6J mice with myelin oligodendrocyte glycoprotein. Total RNA was isolated from spleen, lymph nodes and brain, during two phases and a normal group. Mir193a gene expressions were assessed by qRT-PCR. We also examined the expression of Mir193a in splenocyte and lymphocyte cultures in relapse, remit phases of induced EAE models and normal mice. We found expression level alterations of mir193a during relapse and remit, both *in vitro* and *in vivo*. The results showed a significant increase in expression level of Mir193a in brain samples in remission, compared to relapse phase (p-value=0.0) and normal mice (p-value=0.0). In splenocytes a significant increase of mir193a in remission was observed compared to acute group (p-value=0.021), while in vivo the results were vice versa. In lymph, the relapse samples had significantly increased mir193a compare to remit group (p-value=0.010) and normal samples (p-value=0.017). Lymph nodes *in vitro* results were consistent with *in vivo* results. Mir193a expression pattern was altered during relapse and remit phases of EAE in different tissues. However, the changes depended on the target organ. Interestingly, our results suggest that mir193a may play tissue specific inflammatory or anti-inflammatory roles, therefore, may have remarkable influence in molecular pathogenesis of EAE.

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