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Pathogen recognition receptor pathways are disrupted in the chronic hepatitis-B

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Introduction: Pathogen recognition receptors (PRRs) play key roles in innate immunity against viral infections. It has been also demonstrated that the patients with chronic hepatitis-B (CHB) are unable to eradicate HBV from hepatocytes. It has been hypothesized that innate immunity dysfunction may be considered as a main candidate for specific disrupted immune responses against HBV in the CHB patients. Accordingly, our research team have been evaluated the expression levels of TLRs, TLRs signaling molecules (MYD88, TRIF, IRAK1, TRAF6, IRAK4, TRAF3, NF-κB and IRF7), inflammasomes (NLRP1, NLRP3, NLRC4 and AIM2), inflammasomes downstream molecules (ASC and Caspase-1), MDA5 and RIG-1 in CHB patients.

Material & Methods: This study was performed on 60 CHB patients and 60 healthy controls and the expression levels of the molecules were evaluated by Real-Time PCR technique.

Results: The results demonstrated that expression levels of TLR9 and its intracellular signaling molecules, MDA5, ASC, Caspase-1 and NLRP3 were significantly decreased in PBMCs of CHB patients in comparison to healthy controls.

Conclusion: Based on the results presented here it seems that CHB patients do not express appropriate levels of PRRs in several pathways which may lead to impaired immune responses against HBV infection which is seen in the patients.

Mir193a expression pattern in lymph, spleen and brain samples and cell cultures of experimental autoimmune encephalomyelitis induced mice

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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.