Immunome Res 2016, 12:3(Suppl)
http://dx.doi.org/10.4172/1745-7580.C1.009

The role of macrophages in primary biliary cirrhosis: Novel targets for immune intervention

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Primary biliary cirrhosis (PBC) is a liver specific autoimmune disease (AD) characterized by selective destruction of biliary epithelial cells (BEC) and anti-mitochondrial antibodies (AMA). Numerous studies suggest that PBC ensues from a multi-lineage T and B cell response against the main mitochondrial autoantigen, the E2 component of the pyruvate dehydrogenase (PDC-E2). PBC is often considered a model AD due to the homogeneity of patients and the high specificity of AMA. A major void in the bridge from the loss of tolerance to clinical pathology in PBC is the enigmatic observation that while mitochondria are found in all cells, only small BECs are destroyed. We have recently demonstrated that following apoptosis human BECs translocate PDC-E2 immunologically intact into the apoptotic bodies. Further, we have shown the critical requirement of monocyte derived macrophages (MDMf), from patients with PBC to produce pro-inflammatory cytokines in response to biliary apotopes in the presence of AMAs. ADs affect almost 5% of the western population and have great impact on the quality of life. Our understanding of organ specific AD, including PBC, is compromised by lack of mechanistic understanding as to how specific organ damage occurs. We are addressing a key question for autoimmunity with the overall objective of identifying therapeutic targets for PBC: indeed, strong evidence suggests that impairment in the uptake of apoptotic debris leads to the development of AD.

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The 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 (MS) 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. The *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. The *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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