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Malat-1 LncRNA regulates inflammation and T cell differentiation in an animal model of multiple sclerosis

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Abstract:
BACKGROUND: A growing body of evidence points to the role of long noncoding RNAs (lncRNAs) in the pathogenesis of neurological diseases. Nonetheless, our knowledge of multiple sclerosis-related lncRNAs remains limited. Herein, we investigated the potential role of Malat-1 lncRNA in the context of autoimmune neuroinflammation. **METHODS:** The expression level of Malat-1 was measured in CNS tissues from EAE mice as well as stimulated splenocytes and macrophages using qPCR. To examine the role of Malat-1 in macrophages polarization, Malat-1 siRNA was transfected into primary macrophages followed by M1/M2 macrophage polarization. The expression of M1/M2 markers were then evaluated. Also, the role of the Malat-1 in T cell differentiation was investigated by transfection of CD4+ T cells with Malat-1 siRNA, followed by intracellular cytokine staining. Moreover, effect of Malat-1 downregulation on T cell proliferation was investigated using CFSE staining. **RESULTS:** Expression of Malat-1 was significantly decreased in the spinal cords of EAE mice at days 15 and 25 post disease induction. Stimulated splenocytes showed significant upregulation of Malat-1, whereas expression of Malat-1 in activated macrophages was reduced. Malat-1 downregulation enhanced macrophages polarization towards M1 phenotype. Also, Malat-1 downregulation in activated lymphocytes shifted the pattern of T cell differentiation towards Th1 and Th17 cells while differentiation towards Tregs was decreased. Besides, T cell proliferation was increased following Malat-1 downregulation. **CONCLUSION:** Our data highlight the anti-inflammatory actions of Malat-1 in the context of autoimmune neuroinflammation. Malat-1 influences differentiation of T cells and activation of macrophages, providing potential therapeutic options for controlling inflammation in MS.
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