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SLAMF5 Regulates Myeloid Cells Activation in the Eae Model

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Abstract : Multiple sclerosis (MS) is a chronic neurological disorder characterized by demyelination of the central nervous system (CNS), leading to a wide range of physical and cognitive impairments. Myeloid cells in the CNS, such microglia and border associated macrophage cells, participate in the neuroinflammation in MS. Activation of those cells in MS contributes to the inflammatory response in the CNS and recruitment of immune cells in the this compartment. SLAMF5 is a cell surface receptor that functions as a homophilic adhesion molecule, whose signaling can activate or inhibit leukocyte function. In the current study we followed the expression and function of SLAMF5 in myeloid cells in the CNS and in the periphery in the murine model for MS, the experimental autoimmune encephalomyelitis model (EAE). Our results show that SLAMF5 deficiency or blocking decreases the expression of activation molecules and costimulatory molecules such as MHCII and CD80, resulting in delayed onset and reduced progression of the disease. Moreover, blocking SLAMF5 in peripheral monocytes derived from MS patients and iPSC-derived microglia cells, controls the expression of HLA-DR and CD80. Thus, SLAMF5 is a regulator of myeloid cells function and can serve as a therapeutic target in autoimmune disorders as Multiple Sclerosis.

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