Anglický abstrakt

Experimental autoimmune encephalomyelitis (EAE) is widely accepted as a murine model of human multiple sclerosis autoimmune disease. Murine EAE is usually actively induced by immunization with a suitable myelin antigen. Following immunization, CD4+ T helper lymphocytes Th1 and Th17 accumulate in the nervous tissue and via the production of cytokines, they mediate an inflammatory reaction and the subsequent destruction of myelin.

The main goal of this study was the induction of EAE with clinically observable symptoms and the observation of changes in the counts and phenotypes of cells, mainly NK and T cells. NK cells express a wide range of inhibitory and activation receptors from the C-lectin-like receptor superfamily. The specific ligand of the activating NKR-P1C isoform is still unknown and thus this receptor´s involvement in EAE was also observed. Another goal was the use of medication with regard to the disease progress improvement.

For the purposes of this study, two inbred murine strains with distinct NKR-P1 surface expression were used - the SJL/J strain (expressing inhibitory NKR-P1B) and C57BL/6 (expression activating NKR-P1C).

SJL mice elicited a relapse-remitting of EAE, while C57BL/6 had chronic EAE. Both mouse strains exerted changes in the counts of NK cells and in the expression of their NKR-P1 and NKG-2D receptors. Moreover, interferon beta upregulated NK cell activity and their IL-10 production.