

The dynamics of leukocytes infiltrating CSF in EAE remission during *Heligmosomoides polygyrus* infection in C57BL/6 mice

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Inhibition of coexisting unrelated inflammation is observed during helminth infection. The mechanism of inflammation down-regulation and immunosuppression induced by nematodes remains unknown despite years of study. Infection with the murine intestinal nematode *Heligmosomoides polygyrus* is a widely-used model of immunomodulation. During *H. polygyrus* infection, the symptoms and inflammation associated with ongoing experimental autoimmune encephalomyelitis (EAE), are wholly reduced on the 6th day of infection.

The aim of the study was to determine the dynamics of leukocyte infiltration through the blood-brain barrier (BBB) into inflamed tissue (brain and cerebrospinal fluid – CSF) of mice with EAE, an animal model of multiple sclerosis, infected with nematode.

C57BL/6 female mice were sensitized with myelin oligodendrocyte glycoprotein MOG₃₅₋₅₅ peptide in CFA with *Mycobacterium tuberculosis* H37RA and *Bordetella pertussis* toxin. On the 21st day after sensitization, the animals were infected with 300 larvae stage L3. Examinations were conducted from the 1st to 6th day of infection. Flow cytometry was used to examine leukocytes infiltrating into the CSF. The permeability of the BBB was investigated with various histological and immunological methods. The levels of pro-inflammatory and regulatory cytokines was evaluated. Six days post *H. polygyrus* infection, nervous tissue regeneration was investigated.

Intensified infiltration of leukocytes was observed into the CSF of mice infected with *H. polygyrus*. Changes in BBB permeability were significant and correlated with cell inflow. Differences were also found in regulatory cytokine levels.

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