

Heligmosomoides polygyrus bakeri infection regulates cognitive function in mice

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Due to chronic inflammation, neurodegeneration may progress during the lifespan. It seems that process may be inhibited by immunoregulative mechanisms. Prolonged infection with *H. polygyrus bakeri* is characterized by induction of many factors inhibiting the immune response. The understanding the exact mechanisms behind parasite immunomodulatory and anti-inflammatory properties, is a key for better prevention of nervous tissue destruction and treatment.

The aim of the studies was to evaluate the strength of immunoregulation induced by *H. polygyrus bakeri* infection in mice experimentally programmed for neurodegeneration. The course of systemic inflammation was monitored in the blood of C57BL/6 mice treated with LPS, or F1 generation of C57Bl/6 mice and APP transgenic mice, a mouse model for Alzheimer disease. Three-month-old mice were infected and observed for at least 60 weeks, until resolution of symptoms; and compared with uninfected mice. Changes in the percentage of blood cell population were monitored during the lifespan of mice. The level of cognitive functions linked to progressive neurodegeneration was evaluated in forced swimming test.

In response to LPS treatment, the percentage of neutrophils in the blood decreased and was also low in APP mice. The nematode infection upregulated neutrophil response in elderly and old mice. *H. polygyrus bakeri* infection reduced lymphocyte response and enhanced the level of neutrophils in mice injected with LPS. During prolonged infection, when regular egg production was dropping, the percentage of lymphocytes and neutrophils was stable only in mice treated with LPS or APP.

The results from the forced swimming test showed motor coordination deficits in mice from groups where LPS was injected or from APP group. However, the mice with infection of *H. polygyrus bakeri* without any other sources of inflammation showed longer periods of immobility. Such differences may indicate different immunomodulation mechanisms and its strong dependence on the factor that caused the inflammation.