Summary

„Identification and characterisation of reactive T cells from *Eimeria falciformis* infected mice“

The protozoan parasite *Eimeria falciformis* infects its mouse host and develops in the epithelial cells of the caecum and upper colon. Primary infection of the host by this parasite leads to a stable and specific immunity against subsequent infections. This protection is primarily based on cellular immunity, which is the main subject of this thesis.

Firstly, a parasite-specific proliferation of mesenteric lymph node cells (MLNC) of infected BALB/c-mice was observed in T-cell transformation tests. To determine their possible protective effects *in vivo*, MLNC of infected donor mice were transferred intravenously into naive recipient mice and challenged with 100 oocysts of *E. falciformis*. Recipient mice showed 65% and 74% reduction in oocysts excretion following transfer of donor MLNC from day four and ten post infection (p.i.), respectively. In order to analyse the cell population responsible for the protection, $CD8^+$ T cells of MLNC were separated by MACS (*magnetic cell sorting*) and the effect of this fraction was tested in a transfer experiment. The transfer of the $CD8^+$ T cell population led to a significant reduction of 59-67% in oocyst output.

Due to the high mortality of the recipients of $CD8^+$ depleted MLNC from infected donors, the greatest population of this suspension, the $CD4^+$ T cells, were separated and their effect was examined in a transfer experiment. There were no aggravated clinical symptoms of $CD4^+$ T cell recipient mice compared to the recipients of $CD8^+$ T cells. However, the histological evaluation of pathological changes in the gut showed strong signs of inflammation in the recipients of activated $CD4^+$ T cells, especially in the small intestine. Since *E. falciformis* dwells the caecum and upper colon, these changes in the gut could have been caused by the activity of soluble mediators of inflammation like cytokines. However, the quantification of local and systemic concentrations of the proinflammatory cytokines IFN-γ, TNF-α and IL-6 did not give a clear hint regarding their participation in pathological changes.

To characterise the production of effector T cells and memory cells in an *E. falciformis*-infection, the function of the co-stimulatory receptor CTLA-4 on the surface of T cells was examined in a primary and secondary infection with this parasite. Analysis showed no clear-cut participation of this receptor in the development of protective immune responses against *E. falciformis*.