Genetic influence of *Trypanosoma brucei brucei* infection in mice

The African trypanosomes are zoonotic parasites transmitted by Tse-Tse flies. Two of the three subspecies, *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense*, cause sleeping sickness in humans whereas the third subspecies, *Trypanosoma brucei brucei* is not infective to humans. These parasites are members of Kinetoplastida. Trypanosomes are extracellular parasite witch have complex life cycles involving both insect and mammalian hosts. African trypanosomes after infection penetrate mainly vascularized organs and get into brain where cause serious pathology. Parasite can manipulate with immune system of mammal host in wide spectrum of interactions witch are not clearly understood so far. Discovering of a new immune mechanisms, whitch participite in reaction on african trypanosomes, can reveal some general characteristics of immune system. The results of these studies can help to prepare effective drugs and vaccines against this disease. The best way to observe pathological manifestation and genetical analysis is study on animal models . Study on suitable animal model to find genes which are responsible for control of immune response to *T. brucei* can help us to find homologous genes in humans. It was found that immune responces to african trypanosomes are controled by more than one gene and can result from several combinations of effects of multiple genes that interact in a functional network

To study on genetic control of immune response to *Trypanosoma brucei brucei* infection we use mouse model based on basic strains BALB/c, STS and recombinant congenic strains of CcS/Dem set derived from these basic strains. Each CcS strain contains 12,5% of genome from STS and 87,5% of genome from BALB/c. We have optimised the conditions for intraperitoneal inoculation and we are trying to optimise counting of parasite number in blood. We estimated the viability of choosen mouse strains. The results showed that strain CcS-11 is highly susceptible to *Trypanosoma brucei*. Next we used crossing between CcS-11 and BALB/c for mapping loci controling infection. We infected 169 F_2 hybrids between CcS-11xBALB/c and measured survival time. We typed segments which are different between CcS-11xBALB/c and performed statistical analysis. We obtained 4 loci which controling survival time after *Trypanosoma brucei brucei* infection in mouse (*Tbbr1-4*, *Trypanosoma brucei brucei* response). We also precised *Tbbr2* segment to 2,15 Mbp, contains only 26 genes. Loci *Tbbr3* and *Tbbr4*

interact with each other and contain genes *Cd19* and *Cd5* respectively which are important markers of B-lyphocytes.