Parasitic protists of genera *Trypanosoma* and *Leishmania* are members of *Trypanosomatidae* family. In our studies, we investigated genetic influence on infections caused by these parasites in a mouse model. These diseases are on genetic level controlled by quantitative trait loci (QTLs), when the resulting phenotype is controlled by set of genes with small individual effect. As a mouse model for mapping of QTLs controlling these infections, we used recombinant congenic strains (RCS). Each RCS carry unique set of 12.5% of the genome from donor parental strain on genetic background of other parental strain. For mapping of QTLs controlling infections caused by *Trypanosoma brucei brucei* (*T. b. brucei*) and *Leishmania tropica* (*L. tropica*) and eosinophil infiltration into inguinal lymph nodes after *Leishmania major* (*L. major*) infection, we used RCS from CcS/Dem series, where STS is donor strain and BALB/cHeA is strain of genetic background. First, it was necessary to find suitable model strains for mapping. In all three studies, we selected RCS, which exceeded range of monitored phenotype parameters in comparison with any other tested RCS or parental strains. Mice of RCS CcS-11 showed shorter survival after *T. b. brucei* infection and strain CcS-9 exhibited higher eosinophil infiltration after *L. major* infection. For analysis of genetic control of susceptibility to *L. tropica*, we selected females of the strain CcS-16, which were previously described to have larger lesions and unique chemokine reaction after *L. tropica* infection. In experiments with F_2 hybrids of these strains and background parental strain BALB/cHeA we were able to map four novel loci controlling *T. b. brucei* infection (*Tbbr1-4*), eight loci controlling *L. tropica* infection (*Ltr1-8*) and four loci controlling eosinophil infiltration after *L. major* infection (*Lmr14*, *Lmr15*, *Lmr25* and *Lmr26*). In the segments covering these loci, we found many genes, which were previously described to have a role in investigated infections or eosinophil function but the observed phenotypes can be also controlled by genes with unknown functions in response to these infections, eosinophil function or in immune system in general. We observed strong sex influence in all three response to infections. The difference in survival after *T. b. brucei* infection was more prominent in CcS-11 females than males, larger lesions and unique chemokine reaction was observed only in *L. tropica* infected CcS-16 females and loci *Lmr15* and *Lmr26* controlled eosinophil infiltration after *L. major* infection only in male mice. Many of

newly discovered loci overlap with each other, with previously described loci controlling infections caused by *Trypanosomatidea* family or loci controlling other infections and therefore can share same controlling mechanisms. Next important step in this research will be mechanistic explanation of influence of the discovered loci/genes on disease phenotypes.