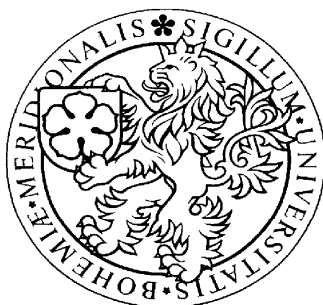


**University of South Bohemia
Faculty of Biological Sciences**



**Effects of interferon gamma and specific polyclonal antibody on
the infection of murine peritoneal macrophages and murine
macrophage cell line PMJ2-R with *Encephalitozoon cuniculi***

RNDr. Thesis

Jiří Jelínek

České Budějovice 2007

Jelínek J. (2007): Effects of interferon gamma and specific polyclonal antibody on the infection of murine peritoneal macrophages and murine macrophage cell line PMJ2-R with *Encephalitozoon cuniculi*. RNDr. Thesis, in English – 5 p., Faculty of Biological Sciences, University of South Bohemia, České Budějovice, Czech Republic.

Annotation: The influence of interferon gamma and specific antibody on the infection of murine peritoneal macrophages and PMJ2-R cell line with *Encephalitozoon cuniculi* was studied. The number of *E. cuniculi* spores was counting in murine peritoneal macrophages and PMJ2-R cell line after lysis. The activity of mice peritoneal macrophages and PMJ2-R cell line was measured by nitrid oxide production.

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I declare that I have worked up this thesis by myself using cited literature.

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Jiří Jelínek

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Effects of interferon gamma and specific polyclonal antibody on the infection of murine peritoneal macrophages and murine macrophage cell line PMJ2-R with *Encephalitozoon cuniculi*, Jiří Jelínek, Jiří Salát, Bohumil Sak and Jan Kopecký, *Folia Parasitologica* 54, 2007.

ABSTRACT

Experimental activation of peritoneal macrophages by interferon gamma (IFN- γ) resulted in the inhibition of *Encephalitozoon cuniculi* replication. However, *E. cuniculi* could replicate either in a non-activated cell line of murine macrophages PMJ2-R or in IFN- γ -activated PMJ2-R cells. Moreover, activation with IFN- γ led to faster replication of *E. cuniculi* in these cells. Opsonisation of *E. cuniculi* spores with anti-*E. cuniculi* polyclonal antibody did not affect *E. cuniculi* replication in both, non-activated and activated murine macrophages. In contrast, opsonisation of *E. cuniculi* spores caused the most effective replication of *E. cuniculi* in activated PMJ2-R cells. However, production of nitric oxide by these cells was significantly more intensive than that in non-activated, infected cells, where the parasite replicated to a much lesser extent. Our results support the hypothesis that *E. cuniculi* uses phagocytosis for the infection of host cells. They also indicate that the mechanism by which spores of *E. cuniculi* are killed by macrophages is not dependent on nitric oxide and they reveal that PMJ2-R cells cannot substitute peritoneal murine macrophages in immunological studies on *E. cuniculi*.