ABSTRACT

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Title of diploma thesis: Entry of bacteria *Mycobacterium bovis* BCG into B lymphocytes

Background: The objective of this work was to evaluate the entry of bacterium *Mycobacterium bovis* BCG into B lymphocytes and the role of selected receptors in this process.

Methods: Peritoneal cell suspensions with unblocked and/or blocked receptors on BALB/c mouse B lymphocytes we infected by bacterium *M. bovis* BCG-GFP unopsonized and/or opsonized by fresh murine serum („complement“) or immune serum („antibodies“). Using flow cytometry we evaluated the entry of bacterium *M. bovis* BCG-GFP into B lymphocytes and their subpopulations B1a, B1b and B2.

Results: *M. bovis* BCG-GFP actively enters into B lymphocytes. Depending on the subpopulation, it most infects B1a, less B1b and at least B2 lymphocytes. Only the subpopulation B2 responds significantly to the opsonization by complement. Opsonization by antibodies had no significant effect on the infection. Entry into CD19+ cells is mediated through the BCR receptor, especially in subpopulations B1a and B1b. Under the opsonized conditions, the CR1/2 complement receptor is applied, and CR3 in the subpopulations B1a and B1b too.

Conclusion: Our results indicate that the BCR receptor alone is sufficient for the recognition and entry of *M. bovis* BCG-GFP into subpopulations B1a and B1b. In the case of opsonization by complement, the complement receptors CR1/2 and CR3 are also used. An interesting result of this work is the marked reaction of the B2 lymphocyte subpopulation to infection by bacterium opsonized by complement, mediated probably mainly via the CR1/2 complement receptor in cooperation with the BCR receptor.

Key words: *Mycobacterium bovis* BCG, B lymphocytes, flow cytometry