ABSTRACT

F. tularensis, an intracellular pathogen, is capable of blocking the signal pathway via TLR4 and MyD88 to transcriptional factor NF-κB in macrophages. Consequently, the expression of genes for proinflammatory cytokines and chemokines (including TNF-α) is reduced. In this study, we investigated if the signal pathway independent of MyD88 is abrogated in macrophages J774 infected with *F. tularensis* LVS. This pathway leads to transcriptional factor IRF-3 and the expression of genes for IP-10 and IFN-β can be influenced. Our results indicate that the signal pathway leading to IRF-3 might be blocked in macrophages infected with *F. tularensis* LVS. We observed lower expression of genes for IP-10 and IFN-β 24 hours after infection.

We also studied if the signal pathway (MyD88-dependent) leading to NF-κB is activated in B-cells A20 infected with *F. tularensis* LVS. Further we investigated if some parallel signal pathway exists also in B-cells. We detected higher expression of the gene for TNF-α in B-cells, indicating that the signal pathway leading to NF-κB might exist here. The parallel signal pathway probably exists in B-cells too, because we observed higher expression of genes for Tbk-1 and IP-10 24 hours after infection.

These data suggest that the interaction of *F. tularensis* LVS with monocyte-macrophage cell line or B-cell line could activate the signal ways differently.