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## Production of Interferon Beta by Toll-like Receptors during Yersinia Pseudotuberculosis Infection


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The plague, a deadly disease caused by the gram-negative coccobacillus *Yersinia pestis*, has caused millions of human deaths around the world. Upon infection, an extreme inflammatory response is triggered that causes further damage to the body. When Toll-like receptors (TLRs) recognize *Y. pestis* bacterium they induce an intracellular signaling pathway that leads to the production of cytokines like Type I Interferon that signal this inflammatory response. Our lab has confirmed that during *Y. pestis* infection the optimal production of Interferon Beta (IFNβ), a subtype of Type 1 Interferon, is dependent on the presence of TLR7 yet not on myeloid differentiation factor 88 (MyD88), its only known signaling adaptor. This indicates a non-canonical TLR7 mechanism in *Y. pestis* infection. We have also observed that wild type mice infected with *Yersinia pseudotuberculosis*, the closest evolutionary relative of *Y. pestis*, have a higher survival rate than *tlr7-/-* mice, indicating that TLR7 may not have the same inflammatory activity during a *Y. pseudotuberculosis* infection. To test the hypothesis that the TLR7 pathway activity in a *Y. pseudotuberculosis* infection is unique from that during a *Y. pestis* infection, wild type and *tlr7-/-* mice were challenged with *Y. pseudotuberculosis* and tissue and blood samples were taken at 5 and 10 days post infection. These samples were analyzed with an ELISA to quantify the levels of INFβ and other cytokines. The results indicate that *Y. pseudotuberculosis* TLR7 are not activated during infection to produce INFβ at the selected time points. Our next step is to test INFβ levels at earlier time points to confirm that there is no immediate TLR7 response and then to confirm which of the well described differences between the two bacteria, *Y. pestis* and *Y. pseudotuberculosis*, is responsible for this difference, revealing more about the non-canonical TLR7 mechanism of *Y. pestis*.