II. ABSTRACT

The bacterium *Francisella tularensis* is a model intracellular pathogen and the causative agent of tularemia. Understanding how this pathogen subverts the immune system to limit inflammation will further our ability to treat infectious and inflammatory diseases. We propose to extend our understanding of a recently identified pathway involving type I interferon, which inhibit IL-17 and neutrophil responses during infection by intracellular pathogens. Our data indirectly suggests that type I interferon stimulates the production of IL-10. Since we have shown that IL-10, an anti-inflammatory cytokine, is required for *F. tularensis* to kill its host, we hypothesize that it is induced as a bacterial strategy to limit immune responses. This proposal first aims to directly assess whether IL-10 inhibits IL-17 and neutrophil responses or acts independently on another inflammatory pathway. Secondly, we aim to identify whether IL-10 production is dependent on type I interferon during infection. Taken together, we hypothesize that type I interferons stimulate the production of interleukin-10, which acts as an anti-inflammatory signal that limits IL-17 and neutrophil responses, and ultimately leads to the death of infected mice.