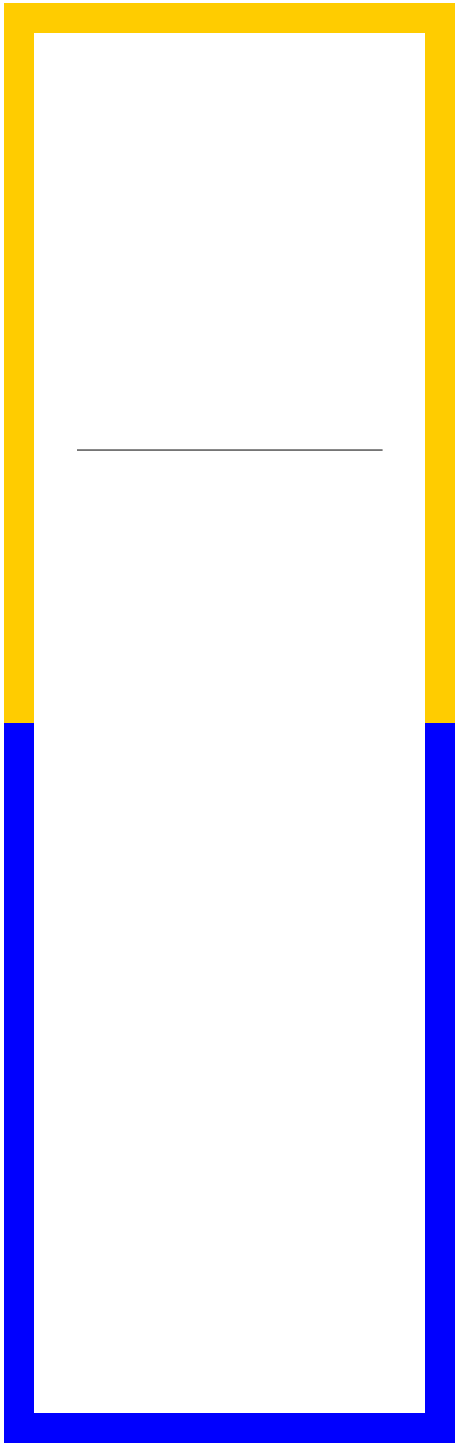
Francisella tularensis is the causative agent of the lethal disease tularemia. Despite decades of research, little is understood about why *F. tularensis* is so virulent. Bacterial outer membrane proteins (OMPs) are involved in various virulence processes, including protein secretion, host cell attachment, and intracellular survival. Many pathogenic bacteria require metals for intracellular survival and OMPs often play important roles in metal binding and uptake. Previous studies identified three *F. tularensis* OMPs that play roles in iron acquisition. We have identified two new proteins, FTT0267 (named *fmvA*, for *Francisella* metal and virulence) and FTT0602c (*fmvB*), which are homologs of those iron acquisition genes and demonstrated that both are upregulated during mouse infections. Based on sequence homology and *in vivo* upregulation, we hypothesized that FmvA and FmvB are OMPs involved in metal acquisition and virulence. Despite sequence homology to previously-characterized iron-acquisition genes, FmvA and FmvB do not appear to be involved iron uptake, as neither *fmvA* nor *fmvB* were upregulated in iron-limiting media and neither Δ*fmvA* nor Δ*fmvB* exhibited growth defects in iron limitation. However, among other metals examined in this study, magnesium-limitation significantly induced *fmvB* expression, Δ*fmvB* was found to express significantly higher levels of lipopolysaccharide (LPS) in magnesium-limiting medium, and increased numbers of surface protrusions were observed on Δ*fmvB* bacteria in magnesium-limiting medium, compared with wild-type *F. tularensis* grown in magnesium-limiting medium. RNA sequencing analysis of Δ*fmvB* revealed the potential



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