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Misregulation of *Burkholderia mallei* strain 10247 results in inappropriate expression of virulence genes and bacterial attenuation

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Abstract

Burkholderia mallei is an intracellular pathogen that has adapted many virulence factors in order to subvert the host immune response and survive within this niche. This pathogen has type III secretion system (T3SS) components that enable it to escape the cell endocytic compartment, a bacterial protein that hijacks host cell actin for its mobilization, and type VI secretion system (T6SS) machinery to block host cell signaling. Previously Losada et al (2010) had reported *B. mallei* 10247 as an attenuated strain in the Syrian hamster model of Glanders infection. In this study we sought to understand the nature of the attenuation observed in *B. mallei* strain 10247. Infection of RAW264.7 macrophages with *B. mallei* 10247 resulted in the inability of the bacteria to escape from the endocytic compartments. Despite this defect, *B. mallei* 10247 was found to overexpress T3SS, T6SS, and capsular genes. We hypothesized that *B. mallei* 10247 elicits a more robust immune response by expressing these antigens inappropriately and sought to determine the nature of the misregulation, since there are no obvious links in the regulation of all of these genes. Confirmation of this hypothesis will identify other, as yet unknown, virulence associated genes that are required for successful subversion of the host immune response and pathogenesis of *B. mallei.*

Biography

Jenifer Ojeda is a postdoctoral research scientist within the Integrated Toxicology Division at USAMRIID. Her current research focuses on elucidating the virulence mechanisms of Category A and B biodefense pathogens *in vivo* and *in vitro*, particularly *Burkholderia* spp. The primary objective of this research is to generate new treatment options for these pathogens using classical approaches (MIC and MBC), high-content imaging, and various -omics.