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Investigating the intracellular survival and virulence of *Burkholderia pseudomallei*

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Complete eradication of *B. pseudomallei*, a CDC-classified class 2 biosafety pathogen, from melioidosis patients is difficult with a high recurrence rate. Intracellular lifecycle of this pathogen has been reported and accepted as the major factor for latent and recrudescing infections in man. Experimental data indicates that the type III secretion system cluster 3 (TTSS3) of *B. pseudomallei* is involved in initiation of the intracellular lifecycle. The T3SS3 of *B. pseudomallei* comprises of three groups of proteins: The needle complex proteins, translocators and secreted effector proteins. This T3SS3 system is therefore able to inject a group of proteins, the effector proteins, from the pathogen directly into the cytosol of the host cell utilising the needle-like protein complex and a group of proteins called translocators, serves the role of translocating the effector proteins into the host cells. In our studies, BipC, one of the putative translocator proteins of TTSS3, was functionally characterized using a mutagenesis approach. The bipC- knockout *B. pseudomallei* mutant strain was constructed and utilized for a spectrum of functional characterization in comparison to their parental strain, K96243. The phenotypes of the strains were investigated through a series of in vitro and in vivo assays. Adherence, invasion and intracellular survival were found to be differentially affected. BipC was also verified to subvert the host actin dynamics as demonstrated by the capability to polymerize actin in vitro. The mean time of death of mice infected with bipC-knockout mutant significantly increased as compared to K96243-infected mice. Altogether, our findings suggest that the protein encoded by the bipC gene may have a role, as a translocator in the pathogenesis of the intracellular lifecycle of *B. pseudomallei* as demonstrated by the attenuated virulence of the bacterium in the in vivo assays.

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