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Burkholderia pseudomallei Bim*C* is required for actin-based motility, intracellular survival and virulence

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The intracellular pathogen *Burkholderia pseudomallei*, the etiological agent of melioidosis in human and various animals, is capable of survival and movement within the cytoplasm of host cells by a process known as actin-based motility. The bacterial factor *BimA* is required for actin-based motility through its direct interaction with actin and by mediating actin polymerization at a single pole of the bacterium to promote movement both within and between cells. However, little is known about the other bacterial proteins required for this process. Here, we have investigated the role of the *bimC* gene (*bpss1491*) which lies immediately upstream of the *bimA* gene (bpss1492) on the *B. pseudomallei* chromosome 2. We have constructed a *B. pseudomallei bimC* deletion mutant and demonstrate that it is defective in intracellular survival in HeLa cells. This defect in intracellular motility in HeLa cells correlates with ablation of plaque and multi-nucleated giant cell (MNGC) formation. These defects in intracellular survival and cell to cell spread are not due to the loss of expression and polar localization of the *BimA* protein inside infected cells; however they do correlate with an inability of the bacteria to recruit and polymerize actin. We also establish a role for *BimC* in virulence of *B. pseudomallei* using a *Galleria mellonella* larvae model of infection. Taken together, our findings indicate that *B. pseudomallei BimC* plays an important role in intracellular behavior and virulence of this emerging pathogen.

Biography

Varintip Srinon has completed her PhD in 2018 from Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand. She is the young researcher at Faculty of Veterinary Science, Mahidol University, Nakhon Pathom, Thailand. She has published more than 4 papers in reputed journals.

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