Mechanistic insights into H. pylori pathogenesis and host response

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Helicobacter pylori, a Gram negative bacterial gastric pathogen, infects ~50% of the world’s population with some progressing to gastric cancer. The indelible link of Helicobacter pylori with gastric diseases coupled with the emergence of multiple-antibiotic resistant H. pylori and high prevalence of gastric cancer calls for attention. However, the mechanistic action of pathogenesis has not been firmly established.

Among the known pathogenic factors of H. pylori reported, cytotoxin associated gene A (CagA) and vacuolating antigen gene A (VacA) have been commonly cited. We have identified two hitherto not described potential pathogenic factors that have shown to be strongly associated with H. pylori pathogenesis and work as “potentiators” to the two virulence factors, CagA and VacA. The first is a surface localized heat shock protein that demonstrates adhesive ability to gastric epithelial cells, colonization capability in BALB/c mice and affects cytoskeleton rearrangement of cells through its interaction with CagA. The second is a membrane bound enzyme that induces generation of ROS, impairs DNA and upregulates IL-8 production.

The dynamics of interaction between the bacterial pathogen and the host, in relation to its 2 potential “effectors” that affect the viability of the host cells will be explored.

Biography
Bow HO completed his PhD at the University of Wales, UK. He is a Professor at the National University of Singapore, an expert in bacterial pathogenesis and host molecular response. He has published >350 journal and conference papers; awarded 19 patents.

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