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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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