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Type IV pili is involved in the pathogenesis of Clostridium difficile in vivo

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Background: *Clostridium difficile* is a Gram-positive spore-forming anaerobic bacterium. It is the leading cause of antibioticassociated diarrhea in nosocomial infection. Recently, Type IV Pili (TFP), a proteinaceous polymer widely studied in many Gram-negative pathogens was discovered to be produced by *Clostridium difficile* and has been reported to promote aggregation, gliding motility and biofilm formation. However, the role that TFP plays in *Clostridium difficile* pathogenesis *in vivo* is still unclear.

Method: TFP structural genes were inactivated using a modified ClosTron targeting system. Antibodies were raised against PilA1, the major component of TFP. For *in vivo* studies, mouse normal flora was disrupted by antibiotic cocktail and then spores were fed orogastrically.

Result: Our preliminary results indicated that TFP mutants were more virulence in mouse model of infection. Furthermore, competition assays and *in vitro* binding assays suggested that TFP mutants outcompeted WT *in vivo* and displayed increase adherence to epithelial cells. Since studies have indicated that the presence of erythromycin resistance gene in mutants could render them become resistant to clindamycin; an antibiotic used during animal studies, we proceed to construct marker-less mutants. Results showed that TFP mutants were still able to induce higher mortality *in vivo*. To further unravel the role of TFP in *Clostridium difficile* pathogenesis.

Conclusion: Our results showed that type IV pili mutants of *Clostridium difficile* caused a more severe disease on mice, which indicates that type IV pili is important in CDI. More works are needed to understand the role of CDI *in vivo*.

Biography

I-Hsiu Huang is an Assistant Professor in the Department of Microbiology and Immunology at the National Cheng Kung University (NCKU). He has worked as a Research Fellow at The University of Texas Health Science Center at Houston and University of Oklahoma Health Sciences Center. He did his PhD at the Oregon State University. His research interests include bacteriology, bacterial genetics and molecular biology.

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