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The study of cAMP receptor protein regulated type-3 fimbriae in Klebsiella pneumoniae

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However, the effect of high blood glucose on the pathogenesis of *Klebsiella pneumoniae* strains remains largely unknown. Bacterial biofilm represents a key virulence determinant in promoting bacterial persistence and resistance to antibiotics. The ability of bacterial adherence to biotic or abiotic substrate is an essential step for biofilm formation. Such adherence can be mediated by bacterial fimbriae, which are also important virulence factors in many bacteria. Type-3 fimbriae, encoded by the mrkABCDF operon genes, are important virulence factors in *Klebsiella pneumoniae* pathogenesis. In enterobacteria, Cyclic AMP Receptor Protein (CRP) plays a vital role as a global regulator. CRP protein regulates several essential bacterial virulence gene expressions, including fimbriae and biofilm formation, responding to intracellular concentration of cAMP. In our preliminary study, we found that different glucose level can regulate the expression of type-3 fimbriae. Also we found that CRP can exactly bind to the putative CRP binding site which located at the promoter region of mrkA (encoding type-3 fimbriae subunit) in *Klebsiella pneumoniae*. Thus, our purpose is to clarify the effect of cAMP receptor protein on type-III fimbriae in *Klebsiella pneumoniae*. Loss of function mutagenesis is an important tool to characterize gene function. Here, we successfully got CRP mutants in *Klebsiella pneumoniae* MGH78578 by Homologous Recombination (HR) using our modified pK18mobsacB suicide plasmid. Our results indicated that CRP can directly regulate the expression of type-3 fimbriae in *Klebsiella pneumoniae*.

Biography

Shih-Wen Cheng is currently pursuing her Masters and his current research interest is in the microbiology field, specifically in *Klebsiella pneumoniae*. She has performed several knockout systems to attain CRP mutants, which included CRISPR/Cas9 system, pKO3 suicide plasmid and pK18mobsacB plasmid. She was successful in attaining the CRP mutants in *Klebsiella pneumoniae* MGH78578 by homologous recombination using modified pK18mobsacB suicide plasmid.

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