Kwan Choi Cheung, J Microb Biochem Technol 2015, 5:4
http://dx.doi.org/10.4172/1948-5948.S1.014

OMICS International
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World Congress and Expo on
Applied Microbiology
August 18-20, 2015 Frankfurt, Germany

The effect of FtsZ inhibitor on the β-lactam resistant activity in methicillin-resistant *Staphylococcus aureus* (MRSA)

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The rapid emergence of antibiotic resistance in *Staphylococcus aureus* (*S. aureus*) leads to the urgent need of alternative drugs to fight against serious bacterial infections caused by *S. aureus*. FtsZ protein is involved in bacterial cell division of most bacteria is therefore an alternative drug target and has been investigated frequently. It even showed synergistic effect when work with β-lactam antibiotic in methicillin-resistant *Staphylococcus aureus* (MRSA). However, the mechanism remains unclear. Previous studies showed that FtsZ inhibitor inhibit bacterial growth. Evidence showed that mode of action of FtsZ inhibitor is bactericidal when combining used with β-lactam antibiotic. This implies the resulting cell lysis and autolysin participation in the drugs action. Effective killing in MRSA reveal the involvement of penicillin binding protein 2A (PBP2A), which has low binding affinity of β-lactam antibiotic. It was thought that FtsZ inhibitor may affect the balance of peptidoglycan hydrolase (autolysin) and peptidoglycan synthase (PBPs). The mechanism and relationship between autolysin, PBP2A and FtsZ protein was therefore first focus on the gene expression in transcriptional level.

Biography

Kwan Choi Cheung has completed her Bachelor from Department of Applied Biology and Chemical Technology, The Hong Kong Polytechnic University. She has experience in R&D, in cosmetic and pharmaceutical industry for several years, and is familiar with cosmetic regulatory affairs. Her interest study is antibiotic resistance mechanism and cell cycle of *Staphylococcus aureus*.

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