

**MOLECULAR CHARACTERIZATION OF RESISTANCE GENES TO ERYTHROMYCIN  
AND CLINDAMYCIN IN CLINICAL ISOLATES OF *STAPHYLOCOCCUS AUREUS* FROM  
UGANDA**

**BY**

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## ABSTRACT

**Background:** Erythromycin (a macrolide) and clindamycin (a lincosamide) are antibiotics that are structurally distinct but related microbiologically because of their similar mode of action of inhibiting protein synthesis by binding to the 50S ribosomal subunits of bacterial cells. They are used to treat infections due *S. aureus* especially methicillin resistant *S. aureus* (MRSA) and vancomycin-resistant staphylococci (VRSA). However, their widespread use and abuse has caused increased resistance in *S. aureus*. Resistance to both of these antimicrobial agents can occur through methylation of their ribosomal target site (encoded by *erm* genes) and by efflux mechanism (mediated by the *msrA* gene). The nature of the underlying genetic mechanism of resistance therefore has clinical implications in the treatment of MRSA, especially if clindamycin is to be considered. Among the Uganda isolates of *S. aureus*, it is not known as to which genetic mechanisms of resistance to macrolides dominate.

**Methods and materials:** This was a cross sectional study conducted in department of medical microbiology of Makerere University from January to May 2014. A total of 255 non duplicate isolates of *S. aureus* from various clinical samples were tested by Kirby Bauer Disk Diffusion and DD test to detect different phenotypes of macrolides, lincosamides and B streptogramins (MLS<sub>B</sub>) resistance. Specific resistance genes were identified using polymerase chain reaction.

**Results:** Phenotypic testing showed that 136 (53.3%) were erythromycin resistant and only 2 (0.08) isolates were constitutively resistant to clindamycin. Forty-three isolates (17%) showed inducible clindamycin resistance phenotype (iMLS<sub>B</sub>), while 91 (35.7 %) isolates were resistant to erythromycin and sensitive to clindamycin (MS phenotype). We also found that 119 (46.7 %) isolates were sensitive to both erythromycin and clindamycin. On PCR, we found that *msrA* (encoding for efflux mechanism) was found in 89 (35%) isolates whereas the *ermC*, *ermA* and *ermB* genes encoding for erythromycin-ribosomal methylase enzyme were detected in 32 (13%), 14 (6%), and 3 (0.01%) isolates respectively.

**Conclusion:** In Uganda, the *msrA* gene is the most dominant genetic mechanism underlying resistance to erythromycin and clindamycin