**Differential Regulation Of Immune Evasion By The *Msa* Gene In Clinical Isolates Of *Staphylococcus Aureus***

*Maria D.S. Basco*[1], *Lindsey N. Shaw*[2], *Mohamed O. Elasri*[1]
[1]*Department of Biological Sciences, The University of Southern Mississippi, Hattiesburg, MS*
[2] *Department of Cell Biology, Microbiology and Molecular Biology, University of South Florida, Tampa, FL*

*S. aureus* is one of the main causes of sepsis usually associated with hospital acquired strains. Reports on the changing epidemiology of staphylococcal infections showed sepsis to be caused by community-acquired strains as well. Remarkably, sepsis caused by CA-MRSA has shown resistance to immunological clearance more than the hospital-acquired ones. Here, we are studying the role of the *msa* gene in regulating immune evasion in the genetic backgrounds of *S. aureus* PFGE types USA300 and USA200. The two strains of *S. aureus* represent a hospital-acquired strain (UAMS-1) and a community-acquired strain (USA300 LAC). Mouse models were used to study sepsis and dissemination. Results of the sepsis model for the *msa* mutant in the UAMS-1 showed the *msa* mutant to be more susceptible to immunological clearance. The *msa* mutant in UAMS-1 showed a 60.3% decrease in survival while in LAC the mutant showed no deterrence. We also found significant fold changes in *clfA, sak, scin,* and *aur* when mutating the *msa* gene. Interestingly, these effects differed in the two genetic backgrounds. The *msa* gene regulates several factors and some of them play specific roles of evasion at the various stages of phagocytic uptake namely, chemotaxis, opsonization, and phagocytosis. Thus, we propose that *msa* could be a regulator of immune evasion and possibly dissemination. Additionally, we show evidence of strain-dependent regulation of immune evasion.