CONCLUSIONS

- Several ncRNAs were screened for their potential targets in *in silico* which indicated multiple mRNA targets encoding virulence factors.
- Two small noncoding RNAs SprX1 and SprB which indicated pathogenicity factors as targets included delta hemolysin, clumping factor B for SprX1 and clumping factor B/A for SprB, were selected for further study.
- The overexpression and disruption of SprX1 positively regulates hemolysin and clumping factor B associated with biofilm formation.
- Conversely, SprB downregulated *clfB/A,* biofilm formation, staphyloxanthin production, while increasing resistance to beta-lactam antibiotics.
- Both *clfB* and *hld* interacted with SprX1 in RNA-RNA interaction studies *in vitro.*
- SprX1 overexpressing strain was more pathogenic than the strain bearing the disruption in mice model of infection. On the contrary, SprB had no significant role in the host.
- These findings revealed that SprX RNA acts as one of the major regulator of pathogenecity factors in the clinical pathogen *S. aureus* Newman.