Overall our results indicate that the impacts of sub-inhibitory chlorhexidine exposure on hospital-associated pathogens should be further investigated in laboratory studies” Bhardwaj et al (2016).

Abstract:

Chlorhexidine is a bisbiguanide antiseptic used for infection control. Vancomycin-resistant E. faecium (VREfm) is among the leading causes of hospital-acquired infections. VREfm may be exposed to chlorhexidine at supra- and sub-inhibitory concentrations as a result of chlorhexidine bathing and chlorhexidine-impregnated central venous catheter use.

We used RNA sequencing to investigate how VREfm responds to chlorhexidine gluconate exposure. Among the 35 genes up-regulated ≥10-fold after 15 minutes exposure to the MIC of chlorhexidine gluconate were those encoding VanA-type vancomycin resistance (vanHAX) and those associated with reduced daptomycin susceptibility (liaXYZ). We confirmed that vanA up-regulation was not strain- or species-specific by querying other VanA-type VRE. VanB-type genes were not induced. The vanH promoter was found to be responsive to sub-inhibitory chlorhexidine gluconate in VREfm, as was production of the VanX protein. Using vanH reporter experiments in Bacillus subtilis and deletion analysis in VREfm, we found that
Chlorhexidine induces VanA-type vancomycin resistance genes in enterococci | 2

this phenomenon is VanR-dependent. Deletion of vanR did not result in increased chlorhexidine susceptibility, demonstrating that vanHAX induction is not protective against chlorhexidine. As expected, VanA-type VRE are more sensitive to ceftriaxone in the presence of sub-MIC chlorhexidine. Unexpectedly, VREfm is more susceptible to vancomycin in the presence of sub-inhibitory chlorhexidine, suggesting that chlorhexidine-induced gene expression changes lead to additional alterations in cell wall synthesis. We conclude that chlorhexidine induces expression of VanA-type vancomycin resistance genes and genes associated with daptomycin non-susceptibility. Overall our results indicate that the impacts of sub-inhibitory chlorhexidine exposure on hospital-associated pathogens should be further investigated in laboratory studies.

Reference:


Thank you to our partners for supporting IVTEAM