**Characterization of *liaX* in *Enterococcus faecalis* lysozyme resistance and biofilm formation**

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*Enterococcus faecalis*, a Gram-positive bacteria inhabiting the human gut, poses a formidable challenge as an opportunistic pathogen due to its capacity to develop antimicrobial resistance and form biofilms. Overcoming the host immune response, including lysozyme-induced killing, is a critical aspect of its pathogenicity. *E. faecalis* cells become more susceptible to lysozyme when the gene encoding the site 2 membrane metalloprotease Eep is disrupted. LiaX, a protein with adhesin and antimicrobial sensing properties, is a key component of the LiaFSR system responsible for sensing and responding to environmental stressors. We found that deletion of *liaX* in the Δ*eep* background represses the loss of *eep*-dependent lysozyme resistance, implicating that *liaX* contributes to the conferral of lysozyme resistance. We hypothesize that deletion of *liaX* in the Δ*eep* background restores lysozyme resistance by remodeling the cell membrane and modifying cell surface net charge. In support of this hypothesis, the Δ*eep* Δ*liaX* strain has increased sensitivity to membrane-targeting detergents and the cationic antimicrobial polymyxin B. Understanding the mechanisms of lysozyme resistance and biofilm formation is important for developing targeted strategies against *E. faecalis* infections. The identification of *liaX* as a pivotal player in these processes opens up novel avenues for interventions, such as disrupting biofilm formation and mitigating antibiotic resistance. Furthermore, recent studies showing the conservation and functional significance of *liaX* across different bacterial pathogens highlights the potential application of our research in understanding antimicrobial resistance mechanisms and designing targeted therapies in other bacteria as well.