Crystallization of phosphomimetic and inactive LiaS in *Enterococcus faecium*

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According to the CDC, antibiotic resistant bacteria kill at least 23,000 people annually. Vancomycin resistant enterococci live in the human gut, but are extremely dangerous to those with compromised immune systems, making it a major cause of hospital acquired infections (HAI). Daptomycin is a last resort antibiotic that is used to treat these infections, but daptomycin nonsusceptible (DNS) strains of bacteria are emerging. In *Enterococci faecium*, antibiotic resistance has been attributed to the LiaFSR gene operon, which organizes the bacterial cell's response to antibiotics that affect membrane integrity. LiaS, the system's sensor, is bifunctional and has a histidine-kinase/HK "off" mode and a phosphatase "on" mode. In the presence of antibiotics and other membrane stressors, LiaS phosphorylates LiaR, the response regulator, in order to transcribe other operons that function in antibiotic resistance. However, it was recently discovered that the mutant $LiaR^{W73C}$, which coevolves with $LiaS^{T120A}$, can induce the dimerization of LiaR by phosphomimicking. Site directed mutagenesis was used to "lock" the protein $LiaS^{T120A}$ into its inactive and phosphomimetic states through an H→A and H→E amino acid substitution in the 164th position, respectively. The protein was then crystallized and the data from the crystal diffraction patterns will be used to solve the structure. Knowing the structure of LiaS could assist in the development of new approaches to increasing the bactericidal effectiveness of antibiotics.