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Abstract:

Metabolism the forbidden fruit of antibiotics

As antibiotic resistance rises, the treatment of serious *Staphylococcus aureus* infections becomes more challenging. To address this problem, pharmaceutical companies and academic researchers tend to focus their efforts on two approaches: developing and/or discovering new antibiotics, and re-purposing human use approved drugs. Metabolomic studies of isogenic daptomycin susceptible and non-susceptible *S. aureus* strain pairs (*Antimicrob. Agents Chemother*. 2015. 59:4226-4238) revealed that daptomycin non-susceptible strains had decreased tricarboxylic acid cycle activity, increased synthesis of pyrimidines and purines, and increased carbon flow to pathways associated with wall teichoic acid and peptidoglycan biosynthesis relative to their daptomycin susceptible counterparts. If these metabolic changes are necessary for the daptomycin non-susceptible phenotype, then will altering the metabolome re-sensitize daptomycin non-susceptible *S. aureus* to daptomycin?

Bio-summary:

Dr. Somerville’s research focus is the elucidation of mechanisms by which bacteria regulate virulence determinants in response to nutrient availability; specifically, my interest is in the function of central metabolism in regulating pathogenesis. Currently, I am an associate professor in the School of Veterinary Medicine and Biomedical Sciences at the University of Nebraska-Lincoln. I earned a Ph.D. degree in Biology from the University of Texas at Dallas under the supervision of Larry Reitzer. Prior to joining UNL, I was a post-doctoral research fellow in the Laboratory of Human Bacterial Pathogenesis at the Rocky Mountain Laboratories, in Hamilton, Montana.