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FOSMIDOMYCIN RESISTANCE IN STAPH SPP. INFECTING VETERINARY ANIMALS

Ishaan Shah

Mentor: Audrey Odom-John

Organisms synthesize isoprenoids (IPP), an essential secondary metabolite, through one of two pathways: the non-mevalonate (MEP) pathway and the mevalonate pathway. While humans use the mevalonate pathway, many microorganisms, including some of the most important pathogens, use the MEP pathway. This makes the MEP pathway an attractive candidate for antibiotic therapy. One antibiotic, fosmidomycin (FSM), competitively inhibits a key enzyme in this pathway. It was recently reported that the *Staphylococcus* spp. that infect household animals, including *S. schleiferi* and *S. pseudintermedius*, utilize the MEP pathway. This suggests that FSM is a strong candidate for the treatment of these *Staphylococcus* spp. However, the mechanism/s by which FSM resistance may be acquired in these strains is unknown. My project will characterize FSM resistance in *S. schleiferi* and *S. pseudintermedius*. For my project, 12 FSM-R strains of *S. pseudintermedius* and *S. schleiferi* were generated and isolated *in vitro*. FSM resistance in these strains was demonstrated using a Minimum Inhibitory Concentration (MIC) assay. In *S. pseudintermedius*, all FSM resistant mutants express a mutation in *GlpT*, a glycerol 3-phosphate transporter responsible for the uptake of FSM. In *S. schleiferi* some of the resistant mutants have mutations in *GlpT*; other mutations of interest are being analyzed as well. Through this research, we will improve our understanding of the MEP pathway in these emerging pathogenic organisms.