Fosmidomycin Resistance in Staph spp. Infecting Veterinary Animals

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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.