An examination of the effect of simvastatin treatment on innate immune responses to *S. aureus* infection

Traci Stankiewicz, Erin Burns, Susan McDowell, and Heather Bruns

Ball State University, Muncie, IN

*Staphylococcus aureus* is one of the most prevalent etiologic agents causing sepsis, a systemic inflammatory response to foreign pathogenic invasion. *S. aureus* infections have become increasingly common. Alternative therapies are needed since prevention of *S. aureus*-induced sepsis requires aggressive medical treatments that are not entirely effective. Simvastatin may be one such treatment. Studies from our collaborator's lab show that simvastatin pre-treatment increases survival of *S. aureus*-infected C57BL/6 mice. The goal of these studies is to examine the effect of simvastatin pre-treatment on the activation of the inflammatory, coagulation, and complement pathways to elicit a mechanism explaining the increased survival of simvastatin-treated, *S. aureus*-infected mice. Thus far, we have examined bacterial clearance and TNFα production, and found that mice pretreated with simvastatin have comparable bacterial clearance and serum TNFα levels when compared to non-treated controls. Establishing the role of simvastatin in these innate immune pathways is important to determining the possibility of simvastatin being used as an adjunctive therapeutic agent for the treatment of sepsis.