1135-VF-1684 **Brittany Bannish*** (`bbannish@uco.edu`). *Exploring Differential Regulation of Blood Clot Degradation.*

Blood clots are physiologically degraded via a biochemical cascade initiated by tissue plasminogen activator (tPA). tPA, which is also used clinically to treat stroke, creates plasmin, the main protein involved in degradation. In this talk, I explore the effects of tPA unbinding and diffusion on clot degradation. I propose that plasmin can "force" tPA to unbind from the clot, which has significant implications for the resulting clot degradation. Using a 3-dimensional stochastic multiscale model, three different regulatory mechanisms are explored when tPA is forced to unbind: 1) tPA is immediately able to rebind to fibrin; 2) tPA is immediately removed from the system; 3) tPA diffuses for some time before being able to rebind to fibrin. I will discuss the contributions of each mechanism in clot degradation, the surprising role of plasmin, and the implications for stroke treatment. (Received September 24, 2017)