1152-92-87 **Marcella Torres**, **Sarah Minucci**, **Shobha Ghosh**, **Rebecca Segal**, **Rebecca Heise** and **Angela Reynolds*** (areynolds2@vcu.edu). *Mathematical modeling of an innate immune response model with macrophage polarization during mechanical ventilation and the early stages of atherosclerosis.*

Macrophages can be activated to a more inflammatory M1 phenotype or to an M2-like phenotype, which promotes the resolution of inflammation. Problems with this phenotypic switching of pro-inflammatory M1 macrophages to an M2 phenotype is essential from appropriate resolution of inflammation can result in a population imbalance that leads to chronic wounds or disease. We have developed a model for the sequential influx of immune cells in the peritoneal cavity in response to a bacterial stimulus that includes macrophage polarization. With this model we are able to reproduce the expected timing of sequential influx of immune cells and mediators in a general inflammatory setting. Sensitivity analysis and numerical simulations were used to explore which dynamics could be targeted to change outcome. We then use this model as a core inflammatory model and illustrate how it can be adapted to various diseases by developing models for both stretch induced lung inflammation during mechanical ventilation and the early stages of atherosclerosis. (Received August 26, 2019)