1145-92-1761   **Rebecca A Segal***, Dept of Mathematics, PO Box 842014, Richmond, VA 23284, and **Marcella Torres**, **Jing Wang**, **Paul J Yannie**, **Shobha Ghosh** and **Angela M Reynolds**. *Development of a mathematical model for the role of inflammation in atherosclerosis.* Preliminary report.

Atherosclerotic cardiovascular disease is a leading cause of morbidity and mortality despite significant advances in lipid management. Complex cellular interactions occur within the artery wall requiring the infiltration/egress of immune cells and lipoproteins within a changing inflammatory milieu and lead to the progression of an atherosclerotic plaque. We developed an ODE model for the influx of immune cells in the peritoneal cavity in response to a bacterial stimulus. Switching of macrophage phenotype from initial pro-inflammatory or M1 phenotype to anti-inflammatory or resolving M2 phenotype is described. The model parameters were calibrated using experimental data. This model is expanded to describe plaque formation arising from inflammatory events. A two-compartment model includes local and systemic dynamics. The local compartment accounts for inflammation at the site the atherosclerotic plaque and progression to foam cells. In the systemic compartment, cells can be activated by LPS to take up lipoprotein derived cholesterol and become foam cells. We use the model to look at the connection between systemic and local measures of M1, M2, N, and pro-and anti-inflammatories. (Received September 24, 2018)