Meeting: 1006, Lubbock, Texas, SS 16A, Special Session on Partial Differential Equation and its Application in Biomedical Study

1006-35-6 **A I Ibragimov**, **C J McNeal** and **L R Ritter*** (lritter@math.tamu.edu), Dept. of Mathematics, Texas A & M University, 3368 TAMU, College Station, TX 77843-3368, and **J R Walton**. *A Mathematical Model of Atherogenesis as an Inflammatory Response.*

We construct a mathematical model of formation of an atherosclerotic lesion based on Russell Ross's paradigm of atherosclerosis as a chronic inflammatory response. This disease results in lesions in the artery that may restrict blood flow, and in critical cases can rupture resulting in heart attack, stroke, or death. When modified low density lipoproteins (oxLDL) enter into the wall of an artery, they can trigger an immune response mediated by chemical signals sent and received by immune and other cells in the vasculature. The presence of oxLDL can also corrupt immune function triggering chronic inflammation. In the construction of our mathematical model, we focus on the inflammatory component of the pathogenesis of cardiovascular disease (CVD). Since this study centers on the interplay between chemical and cellular species, we employ the model of chemotaxis given by E. Keller and L. Segel (1970), and present our model as a coupled system of nonlinear reaction diffusion equations describing the state of the species involved in the disease process. We perform some numerical analyses and demostrate that our model captures observed features of CVD such as localization of immune cells, build up of lipids and debris, and isolation of a lesion by smooth muscle cells. (Received October 07, 2004)