1014-92-1404 **Laura R Ritter*** (lritter@math.tamu.edu), Texas A & M University, Dept. of Mathematics, 3368 TAMU, College Station, TX 77843-0001, and **Akif I Ibragimov**, **Catherine J McNeal** and **Jay R Walton**. *A mathematical framework for the modeling of atherosclerosis.*

We consider the onset of atherosclerosis as an inflammatory response in the presence of injury to the endothelial layer and excess levels of oxidized low density lipoproteins in the subendothelium. The bio-chemical signaling that occurs during injury or invasion by foreign bodies, and the immune system response is viewed as key to the initiation of a diseased state. We propose a mathematical framework to model initiation (atherogenesis) and development of the disease, based in part on the classical model of chemotaxis given by Keller and Segel. We present a model as a coupled system of nonlinear reaction diffusion equations describing the state of the species involved in the disease process. Numerical analyses demostrate that our modeling captures certain observed features of cardiovascular disease such as the localization of immune cells, the build up of lipids and debris, and the isolation of a lesion by smooth muscle cells. A classical steady state analysis also suggests that intitiation of disease can be viewed as the result of instability. A stability criterion, and its biomedical implications, is discussed. Finally, a spatially nonhomogeous steady state analysis is considered. (Received September 28, 2005)