ABSTRACT:

Atherosclerotic plaques are fatty deposits that grow mainly in arteries and develop as a result of a chronic inflammatory response. Although plaques are frequently thought of as fatty deposits with little or no internal organization, they are actually commonly characterized according to their composition and morphology. In this talk, I will present models for two types of plaque: thin-cap fibroatheromas (TCFAs) and pathological intimal thickenings (PITs).

TCFAs are characterized by inflammation and the presence of necrotic cores. By solving coupled reaction-diffusion equations for macrophages and dead cells, we explore the joint effects of hypoxic cell death and chemoattraction to oxidized low-density lipoprotein (Ox-LDL), a molecule that is strongly linked to atherosclerosis. The model predicts cores that have approximately the right size and shape when compared to ultrasound images. Normal mode analysis and calculation of the smallest eigenvalue enable us to compute the formation times of the core. An asymptotic analysis reveals that the distribution of Ox-LDL within the plaque determines not only the placement and size of cores, but their time of formation.

PITs are characterized by the absence of endothelial cells and negative remodeling whereby the vessel lumen decreases in size. I will present some work in progress on PIT, described as a free-boundary problem. The model couples the diffusion of Platelet-Derived Growth Factor, governed by a Helmholtz equation, to cell migration and proliferation. The predictions are compared with data from animal studies.