Coronary artery disease (CAD) is considered as the leading cause of death in the developed world and is a result of plaque buildup (Atherosclerosis) in the coronary arteries, which interrupts blood flow. Atherosclerotic plaque develops silently over a prolonged period of time, through a highly complicated inflammatory process that includes many factors, molecules and reactions. Although atherosclerosis is a systemic disease, there are two common cases that their manifestations are acute. In the first case, the stenosis gets severe as a result of the plaque growth (more than 80% blockage), until it blocks the blood flow. The second case, appears without any advance symptoms, is caused by increased stresses on a vulnerable plaque (usually less than 50% blockage) which lead to plaque rupture. Plaque rupture with superimposed thrombosis, similar to a severe stenosis, is the cause of acute coronary syndromes such as myocardial infarction (MI) and stroke. Hence, coupled hemodynamic, structural stress analyses and macromolecules’ reactions are pivotal to understanding plaque formation, stability and vulnerability to rupture.

Studies aimed at understanding atherosclerosis need to be broad in scope and integrative in nature. The appropriate framework in which to consider emergent dynamical behavior of this type is mathematical and computational modeling. Researches on models and computational simulations that describe the phenomena are starting to proliferate, due to its obvious significance to public health. There are greatly varying degrees of complexity in these computational studies depending on the number of species considered, number of constant parameters, the development of the equations proposed, the reactions’ terms used and the dimensional complexity.

The current study is of simplified approaches for modeling the initiation and progression of atherosclerotic plaque, through ignoring plaque spatial structure and taking a population-type manner. Since the geometry of a blood vessel is patient-specific and very complex, we believe that a non-spatial model, allows us to include many parameters in a simple and elegant way, perform stability analyses and investigate many different cases without the necessity of computer resources. This way, in addition to critically reviewing various mathematical models, we aim to describe different aspects of the mechanisms that drive atherosclerosis.