Effect of HDL ("good cholesterol") in a model of early atherosclerosis

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The first event that leads to atherosclerosis is an injury to the endolthelium, which is the lining of an artery. LDL ("bad cholesterol") penetrates this injury and enters the intima, the top layer of the artery wall. At the same time the injured endothelium sends out signals that attract macrophages (immune cells) that also enter the intima and engulf the LDL and become fat-laden foam cells. We have a working mathematical model for this process and this model predicts that increasing the number of macrophages in the blood stream, when there are high levels of LDL, leads to a discontinuous increase in the number of macrophages in the intima via a fold bifurcation.

In this project we will add HDL to the model and look at its effects on the accumulation of foam cells. HDL is able to remove fat from foam cells so that these cells return to being macrophages and can resume engulfing LDL. We will use AUTO bifurcation software to look at the steady states that the model predicts and find out how the presence of HDL effects the bifurcation behaviour of the model and whether the model predicts sudden changes in LDL, foam cell or macrophage levels as macrophage availability changes.

Suitable for: Students who have completed a course on PDE's, enjoy finding numerical solutions (i.e. computing) and who are willing to learn about atherosclerosis in some detail.