Abstract
Platelets play an essential role in blood clotting; they adhere to damaged tissue and release chemicals that activate other platelets. Yet in order to adhere, platelets, which account for only 0.3% of the blood’s volume, must first come into contact with the injured vessel wall. Fortunately, fluid dynamics has solved this problem. Under arterial and arteriolar flow conditions, platelets have an enhanced concentration near blood vessel walls. This non-uniform cell distribution depends on the fluid dynamics of blood as a heterogeneous medium; no such effect occurs in a Newtonian fluid.

Although lateral platelet motion has been well documented, the fluid dynamics are poorly understood. We use a parallelized lattice Boltzmann-immersed boundary method to solve the flow dynamics of red cells and platelets in a 2D vessel with no-slip boundary conditions. Using this method we analyze the influence of hematocrit and shear rate on lateral platelet motion. Insight gained from this work could lead to significant improvements to current models for platelet adhesion where the presence of red blood cells is neglected due to computational intensity.

Lateral platelet motion
Blood is a heterogeneous medium composed primarily of red blood cells, plasma and platelets. Platelets have a non-uniform concentration that is highly localized near vessel walls where they repair vessel damage. Experiments by Eckstein et al. [3] show that this concentration profile is highly sensitive to both shear rate and hematocrit.

To simulate this effect in models of platelet adhesion, Eckstein et al. [2] suggested continuum and stochastic drift-diffusion models for platelet motion with constant diffusion and a drift defined by the equilibrium platelet concentration taken from experiments that pushes platelets toward the wall.

Immersed boundary-lattice Boltzmann method
The lattice Boltzmann equations govern the behavior of the fluid in our simulations. These equations track particle distribution functions \( f(x, c, t) \) that live at each Eulerian lattice node \( x \) at time \( t \). Each PDF is allowed to move along a set of discretized velocity vectors, \( c \).

The equations governing the particle distribution functions in the presence of an immersed boundary force are

\[
\frac{df(x, c, t)}{dt} = \nabla \cdot (\mathbf{F} f(x, c, t)) + \eta f(x, c, t),
\]

where \( r \) is the relaxation parameter, \( c \) is the particle speed, and \( f^{eq} \) is the equilibrium distribution. The macroscopic quantities \( \rho(x, t), \mu(x, t) \) and \( u(x, t) \) are the density and macroscopic fluid velocity. These quantities can be calculated from the particle distribution functions by

\[
\rho(x, t) = \int c f(x, c, t) \, dc, \quad \mu(x, t) = \int c^2 f(x, c, t) \, dc - \frac{1}{2} \rho(x, t) u(x, t), \quad u(x, t) = \int c f(x, c, t) \, dc.
\]

In the limit that the lattice spacing approaches zero and the particle speed approaches infinity, the lattice Boltzmann equations approximate the Navier Stokes equations where the fluid pressure and viscosity are

\[
\rho_{eq}(x, t) = \frac{1}{3} \mu_{eq}(x, t), \quad \mu = \frac{1}{2} \rho_{eq}(x, t) \mu_{eq}(x, t).
\]

The immersed boundary method is a way of coupling fluid dynamics to the mechanics of some elastic boundary location within the fluid. Fluid lies on Eulerian grid \( x \) and the boundary lives on a Lagrangian grid \( X(x, t) \). An external force is imposed on the fluid by the presence of the immersed boundary object. The boundary moves with the fluid velocity, and the force felt by fluid is related to the force felt by the boundary.

\[
F_{\text{boundary}} = \int F_{\text{fluid}}(x, X(x, t)) \, dx, \quad F_{\text{fluid}} = \int \left( \frac{\partial \rho_{eq}(x, t)}{\partial x} - \frac{\partial \mu_{eq}(x, t)}{\partial t} \right) \, dx.
\]

To model the behavior of one-dimensional cellular membranes, we use the following force equation

\[
F_{\text{boundary}} \left( \frac{dx}{dt}, X(x, t) \right) = \int \left( \frac{\partial \rho_{eq}(x, t)}{\partial x} - \frac{\partial \mu_{eq}(x, t)}{\partial t} \right) \, dx.
\]

\( x(t) \) is the instantaneous curvature and \( r_{eq} \) is the curvature of minimum energy. Tension is both tangent to plane (due to stretching) and normal to plane (due to bending). For in-plane tension we use a one-dimensional reduction of the Skalak membrane law

\[
T_{\text{membrane}} = G X^2 (1 + \lambda^2 X^2)^{-1} + \frac{1}{2} \mu (X^2 + 3),
\]

where \( \lambda \) is the principal stretch ratio.

Numerical experiments
Below are simulations of red blood cells and platelets under pressure-driven flow for (a) 20% hematocrit and (b) 40% hematocrit.

Conclusions and Future directions
Our preliminary results show that lateral platelet motion is caused by interactions with red blood cells. We also have reason to believe that enhanced diffusion alone is not enough to generate this motion; that a barrier may exist at the edge of the red blood cell layer and this barrier effectively causes a net “drift” toward the vessel wall.

In the future, we hope to:
- create stochastic differential equation model of platelet transport
- characterize platelet motion around a clot

References