Motivation

- Valeri and coworkers (2001) studied surgical patients
- Patients with low red blood cell (RBC) concentrations experienced significantly longer bleeding times
- Patients with low platelet concentrations experienced no change in bleeding time
- Zhao, Narsimhan, and Shaqfeh built a large blood simulation
- RBC deformability causes accumulation in the center of the channel
- There is an RBC-free region near the wall termed the Fahraeus-Lindqvist layer
- Platelets accumulate in the F-L layer
- After being pushed to the outside of the channel, how do platelets attach to damaged endothelial cells?

Methods

- Stokes flow simulation
  - Single-layer boundary element method
  - Wall bounded Green’s function based on Blake’s Stokeslet
- Model
  - Platelets – rigid oblate ellipsoids
  - Ligand-receptor bonds – Hookean springs
  - Spring dynamics – first order rate kinetics

Adhesion process

- After trauma, damaged endothelial cells express collagen
- vWF (large protein in blood) anchors to collagen and then recruits passing platelets
- Platelets transiently adhere to vWF through the GPIb receptor, rolling along the surface until firm adhesion occurs, mediated by integrin αIIbβ3

Large Simulation Data Mining

- Platelet collisions with red blood cells lead to shear-induced diffusion
- Factor of 10 larger than Brownian motion
- Modeled as an autoregressive process with appropriate velocity fluctuations and autocorrelation

Experiments

- Inverted microscope setup watching platelets roll on collagen surface
- Output platelet trajectories
  - Trajectory length
  - Trajectory speed
  - Firm vs transient adhesion

References

