

Abstract Submitted  
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**In-vitro measurement and modelling of shear-induced platelet margination and adhesion in channel flows** QIN M. QI, Stanford University, IRENE OGLESBY, JONATHAN COWMAN, Royal College of Surgeons in Ireland, ANTONIO J. RICCO, Dublin City University, DERMOT KENNY, Royal College of Surgeons in Ireland, ERIC S.G. SHAQFEH, Stanford University — Blood coagulation is initiated by GPIb and GPIIb/IIIa receptors on the platelet surface binding with von Willebrand factors tethered on the vascular wall. This process occurs much faster in the presence of flow shear than in the quiescent fluid. First of all, the near-wall platelet concentration in flowing blood increases significantly. This phenomenon, commonly referred to as platelet margination, is due to shear-induced hydrodynamic interactions between red blood cells and platelets. Flow shear also manifests itself in affecting the reaction kinetics of receptor-ligand binding. The breaking and formation of multiple bonds on the platelet surface result in the translocating motion of platelets rolling close to the vascular wall. To date, a fundamental understanding of how fluid mechanics relate the bond-level kinetics to the platelet-level dynamics is very limited. In this talk, we investigate platelet adhesion under physiological shear rates using both microfluidic experiments and multi-scale modeling. Our model, (based on existing single molecule measurements and hydrodynamics of blood at zero Reynolds number) shows good agreement with experimental results. We discuss the roles of red blood cell volume fraction (hematocrit), shear rate, receptor densities in the dynamics of platelet adhesion. These findings also provide implications for how platelet defects and abnormal flow conditions influence hemostasis and thrombosis.

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