Abstract Submitted for the DFD12 Meeting of The American Physical Society

Mechanism of vaso-occlusion in sickle cell anemia¹ HUAN LEI, GEORGE KARNIADAKIS, Brown University — Vaso-occlusion crisis is one of the key hallmark of sickle cell anemia. While early studies suggested that the crisis is caused by blockage of a single elongated cell, recent experimental investigations indicate that vaso-occlusion is a complex process triggered by adhesive interactions among different cell groups in multiple stages. Based on dissipative particle dynamics, a multi-scale model for the sickle red blood cells (SS-RBCs), accounting for diversity in both shapes and cell rigidities, is developed to investigate the mechanism of vaso-occlusion crisis. Using this model, the adhesive dynamics of single SS-RBC was investigated in arterioles. Simulation results indicate that the different cell groups (deformable SS2 RBCs, rigid SS4 RBCs, leukocytes, etc.) exhibit heterogeneous adhesive behavior due to the different cell morphologies and membrane rigidities. We further simulate the tube flow of SS-RBC suspensions with different cell fractions. The more adhesive SS2 cells interact with the vascular endothelium and further trap rigid SS4 cells, resulting in vaso-occlusion in vessels less than $15\mu m$. Under inflammation, adherent leukocytes may also trap SS4 cells, resulting in vasoocclusion in even larger vessels.

 $^1\mathrm{This}$ work was supported by the NSF grant CBET-0852948 and the NIH grant R01HL094270.

Huan Lei Brown University

Date submitted: 26 Jul 2012

Electronic form version 1.4