Abstract Submitted
for the MAR12 Meeting of
The American Physical Society

Biomechanics of the endothelium substrate influences leukocyte transmigration[1] KIMBERLY STROKA, HELIM ARANDA-ESPINOZA, Fischell Department of Bioengineering, University of Maryland — The effects of shear flow and cytokines on leukocyte transmigration are well understood. However, the effects of substrate stiffness on transmigration remain unexplored. We have developed an in vitro model that allows us to study leukocyte transmigration as a function of varying physiological substrate stiffness. Interestingly, leukocyte transmigration increased with increasing substrate stiffness below the endothelium. intercellular adhesion molecule-1 expression, stiffness, cytoskeletal arrangement, morphology, and cell-substrate adhesion could not account for the dependence of transmigration on substrate stiffness. We also explored the role of cell contraction and observed that (1) neutrophil transmigration caused large hole formation in monolayers on stiff substrates. (2) Neutrophil transmigration on soft substrates was increased by decreasing cell-cell adhesion. (3) Inhibition of myosin light chain kinase normalized the effects of substrate stiffness by reducing cell contraction on stiff substrates. These results demonstrate that neutrophil transmigration is regulated by MLCK-mediated generation of gaps at cell borders through substrate stiffness-dependent endothelial cell contraction.

[1]Authors acknowledge support from NSF CMMI-0643783

Helim Aranda-Espinoza
Fischell Department of Bioengineering, University of Maryland

Date submitted: 10 Nov 2011 Electronic form version 1.4