Abstract Submitted
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Rheology of platelets at late stages of activation and their role in thrombo-inflammatory responses[1] DAVID BARK JR., Department of Mechanical Engineering, School of Biomedical Engineering, Colorado State University, KATRINA ASHWORTH, Department of Pediatrics, University of Colorado Anschutz Medical Campus, YUPING YUAN, Charles Perkins Centre, The University of Sydney, JORGE DIPAOLA, Department of Pediatrics, University of Colorado Anschutz Medical Campus, SHAUN JACKSON, Charles Perkins Centre, The University of Sydney — Thrombo-inflammatory responses can lead to death and can be found in reperfusion injury, deep vein thrombosis, and organ transplantation. Key characteristics include microvascular thrombi and intravascular leukocyte aggregation. We demonstrate that the heterotypic interaction of platelets and leukocytes is highly dependent on flow and platelet rheology. For this work, we visualize platelets at various stages of activation and their interactions with leukocytes in microfluidic flow chambers at various wall shear rates. We further investigate the binding mechanisms supporting the interactions by using blood from a Nbeal2^{KO} mouse. Through this work, we find that as platelets reach late stages of activation, they lose their structural integrity, resulting a membrane shell. Through flow experiments, we find that the shell increasingly deforms as the wall shear rate increases to 28,800 s^{-1}, with little link between wall shear rate and membrane rupture. However, when quantifying membrane tension, rupture occurs consistently at 20 pN/μm. By further exposing P-selectin on their surface, platelets can support leukocyte binding at a shear rate of 200 s^{-1} or less, supporting membrane tension that exceeds the rupture limit, due to drag forces associated with leukocyte size.

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