

Supplemental Materials: Development of a Glycosaminoglycan Derived, Selectin Targeting Anti-Adhesive Coating to Treat Endothelial Cell Dysfunction

James R. Wodicka ^{1,2}, Andrea M. Chambers ¹, Gurneet S. Sangha ¹, Craig J. Goergen ¹ and Alyssa Panitch ^{1,3,*}

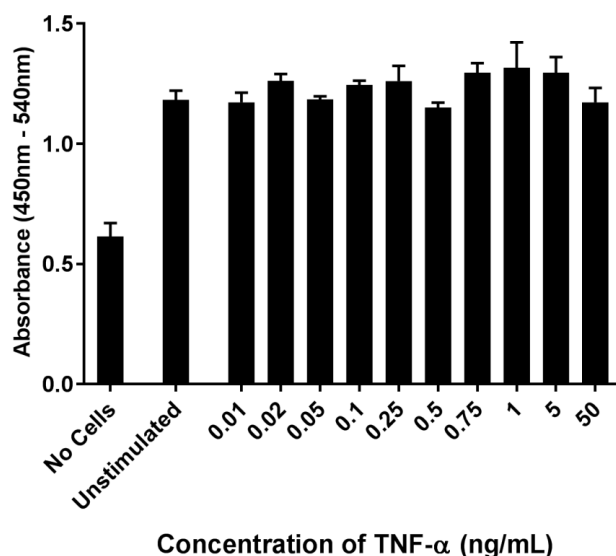


Figure S1. Selectin expression on ECs when stimulated with varying concentrations of TNF- α . Expression was quantified using primary anti-E-selectin and secondary HRP-conjugated antibodies. Selectin levels did not change when stimulated with TNF- α . $n = 3$; $p < 0.05$.

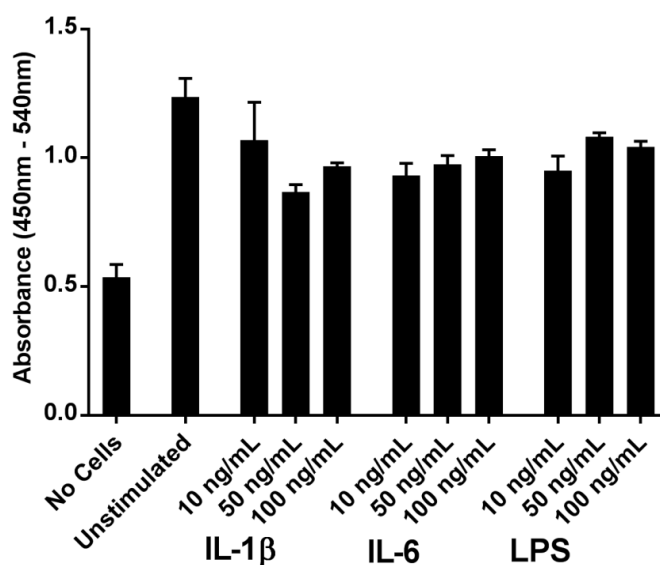


Figure S2. Selectin expression on ECs when stimulated with varying concentrations of IL-1 β , IL-6 and LPS. Expression was quantified using primary anti-E-selectin and secondary HRP-conjugated antibodies. Proinflammatory stimuli failed to increase selectin expression on the EC surface. $n = 3$; $p < 0.05$.

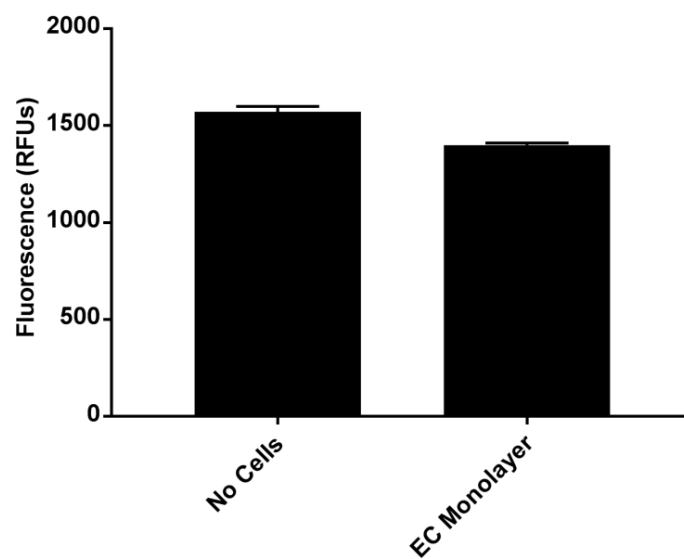


Figure S3. EC monolayer permeability in unstimulated conditions. RITC-dextran was added to the upper chamber of Transwells with and without an EC monolayer. Following incubation, a sample from the lower chamber was collected and fluorescence (Ex: 520 nm; Em: 590 nm) was recorded. Despite ECs present, a majority of the RITC-dextran was able to pass through the Transwell, indicating that the monolayer was not fully intact. $n = 2$; $p < 0.05$.