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White blood cell deformation and firm adhesion ALEX SZATMARY, CHARLES EGGLETON, University of Maryland, Baltimore County — For a white blood cell (WBC) to arrive at infection sites, it forms chemical attachments with activated endothelial cells. First, it bonds with P-selectin, which holds it to the wall, but weakly; this allows the WBC to roll under the shear flow of the blood around it. Later, the WBCs bond with the stronger intracellular adhesion molecule-1 (ICAM-1); it is these ICAM bonds that allow the WBCs to fully resist the flow and stop rolling, allowing them to crawl through the endothelial wall. We model this numerically. Our model uses the immersed boundary method to represent the interaction of the shear flow with the deformable cell membrane. Receptors are on the tips of microvilli-little fingers sticking off of the cell membrane. The microvilli also deform. The receptors stochastically form and break bonds with molecules on the wall. Using this method, the history of each microvillus and its bonds can be found, as well as the distribution of the adhesion traction forces and how all of these vary with the deformability of the white blood cell. At higher shear rates, the white blood cell membrane deforms more, increasing its contact area with the surface; this effect is larger for softer membranes. We investigate how the deformability of the WBC affects the ease with which it forms firm adhesion.

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