

Abstract Submitted  
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**Theoretical modeling of the catch-slip bond transition in biological adhesion** KIM GUNNERSON, YURIY PEREVERZEV, OLEG PREZHDO, University of Washington — The mechanism by which leukocytes leave the blood stream and enter inflamed tissue is called extravasation. This process is facilitated by the ability of selectin proteins, produced by the endothelial cells of blood vessels, to form transient bonds with the leukocytes. In the case of P-selectin, the protein bonds with P-selectin glycoprotein ligands (PSGL-1) produced by the leukocyte. Recent atomic force microscopy and flow chamber analyses of the binding of P-selectin to PSGL-1 provide evidence for an unusual biphasic catch-bond/slip-bond behavior in response to the strength of exerted force. This biphasic process is not well-understood. There are several theoretical models for describing this phenomenon. These models use different profiles for potential energy landscapes and how they change under forces. We are exploring these changes using molecular dynamics. We will present a simple theoretical model as well as share some of our early MD results for describing this phenomenon.

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