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**Bond tilting and sliding friction in a model of cell adhesion** SYLVAIN REBOUX, GILES RICHARDSON, OLIVER JENSEN, University of Nottingham — As a simple theoretical model of a cell adhering to a biological interface, we consider a rigid sphere moving in a viscous shear flow near a wall. Adhesion forces arise through intermolecular bonds between receptors on the cell and their ligands on the wall, which form flexible tethers that can stretch and tilt as the base of the cell moves past the wall; binding kinetics is assumed to follow a standard model for slip bonds. Our model reveals three distinct types of motion: either bonds accumulate at the peeling edge and slow down the cell almost to a halt; or bonds adhere strongly, but without creating any significant torque, and the cell tank-treads over the wall without slipping; or the cell moves near its free-stream speed with bonds providing weak frictional resistance to sliding. Under realistic conditions, the model predicts bistability among these three states, implying that at critical shear rates the system can switch abruptly between firm adhesion, tank-treading and free sliding. The model suggests that sliding friction arising through bond tilting may play a significant dynamical role in cell–adhesion applications such as neutrophil rolling and bacterial colonization under flow.

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