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**Heritable adhesion geometries and mechanosensing of surfaces by biofilm-forming bacteria** VERNITA GORDON, BENJAMIN COOLEY, CHRIS RODESNEY, NUMA DHAMANI, The University of Texas at Austin — Biofilms are dense, interacting communities of single-celled organisms that are bound to each other with a self-produced polymer matrix. Biofilms have devastating clinical impact as they increase resistance to antibiotics and the immune system as well as the production of virulence factors that damage the host. Here we examine effects very early in biofilm development, when the infection is still in a stage of a few cells not yet characterized by high biofilm densities. *Pseudomonas aeruginosa*, an opportunistic human pathogen, produces multiple extracellular polysaccharides that form the biofilm's structuring matrix. We have recently shown that the two primary polysaccharides, Pel and Psl, have distinct roles in controlling the mechanics of single-cell adhesion to a surface – Psl dominates adhesion to the surface, and Pel makes the bacterium lie down flat (Cooley *et al.*, 2013 Soft Matter). Here, we show that expressing Pel alters the symmetry of Psl's distribution on the surface of rod-shaped *Pseudomonas*. We also show that expressing Pel decreases the work of detachment from the surface. It seems paradoxical that a biofilm-forming organism should pay the cost of maintaining and making a gene product that reduces the energy input required to detach it from a surface. Therefore, we probe the possibility that a flat-lying bacteria may better sense a solid surface and change its signaling state accordingly.

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