Attachment to a surface triggers intracellular signaling to change gene expression from the planktonic to the biofilm phenotype. *Pseudomonas aeruginosa* has long been known that intracellular levels of the signal cyclic-di-GMP increase upon surface adhesion and that increased cyclic-di-GMP levels are required to begin biofilm development. However, what cue is sensed to notify bacteria that they are attached to the surface has not been known. Here, we show that mechanical shear acts as a cue for surface adhesion and activates cyclic-di-GMP signaling. The magnitude of the shear force, and thereby the corresponding activation of cyclic-di-GMP signaling, can be adjusted both by varying the strength of the adhesion that binds bacteria to the surface and by varying the rate of fluid flow over surface-bound bacteria. We identify specific protein structures as important elements in the mechanosensory process. An analytic model that accounts for the feedback between mechanosensors, cyclic-di-GMP signaling, and production of adhesive polysaccharides describes our data well. It is well-known that eukaryotic cells can sense and respond to many types of mechanical inputs. In contrast, very little is known about how prokaryotes may respond to mechanical inputs. Here, we show that bacteria can sense shear and can respond by initiating biofilms. This is an important advance in fundamental microbiology and mechanobiology. Biofilms are difficult to prevent using extant approaches. Our knowledge points the way to a hitherto undeveloped type of antibiofilm surface that thwarts mechanosensing and thereby prevents bacteria from "knowing" that they are attached to a surface, activating cyclic-di-GMP signaling, and forming a biofilm.