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Modulation of membrane mechanical properties by Sar1, a vesicle trafficking protein.

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The trafficking of cargo in cells involves dramatic changes in membrane shape and topology. Though trafficking is widely studied and the identities and interactions of the responsible proteins are well mapped, remarkably little is known about the mechanics involved. We focus on Sar1, the key regulator of the coat protein complex II (COPII) family that ferries newly synthesized proteins from the ER to the Golgi. Sar1 is the only member of the COPII coat that interacts directly with the ER lipid bilayer membrane. It has an amphipathic N-terminal helix; when Sar1 is GTP-bound, the helix is exposed and the hydrophobic hemi-cylinder can insert into the bilayer. To investigate whether Sar1 has a role beyond merely localizing the other COPII proteins, we directly measure the force involved in membrane deformation as a function of its presence or absence, using optically trapped microspheres to pull tethers from lipid membranes whose composition and large surface area mimic the composition and geometry of the ER. Tether measurements allow extraction of the membrane bending modulus, the parameter that governs the energetics of deformation. We find that the bending modulus measured in the presence of Sar1 with a non-hydrolyzable GTP analogue is half that measured without Sar1 or with Sar1-GDP. These results reveal a paradigm-altering insight into COPII trafficking: Sar1 actively alters the material properties of the membranes it binds to, lowering the energetic cost of curvature generation.