MAR14-2013-020621

Abstract for an Invited Paper
for the MAR14 Meeting of
the American Physical Society

Membrane Bending by Protein Crowding

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From endosomes and synaptic vesicles to the cristae of the mitochondria and the annulus of the nuclear pore, highly curved membranes are fundamental to the structure and physiology of living cells. The established view is that specific families of proteins are able to bend membranes by binding to them. For example, inherently curved proteins are thought to impose their structure on the membrane surface, while membrane-binding proteins with hydrophobic motifs are thought to insert into the membrane like wedges, driving curvature. However, computational models have recently revealed that these mechanisms would require specialized membrane-bending proteins to occupy nearly 100% of a curved membrane surface, an improbable physiological situation given the immense density and diversity of membrane-bound proteins, and the low expression levels of these specialized proteins within curved regions of the membrane. *How then does curvature arise within the complex and crowded environment of cellular membranes?* Our recent work using proteins involved in clathrin-mediated endocytosis, as well as engineered protein-lipid interactions, has suggested a *new hypothesis* - that *lateral pressure generated by collisions between membrane-bound proteins can drive membrane bending.* Specifically, by correlating membrane bending with quantitative optical measurements of protein density on synthetic membrane surfaces and simple physical models of collisions among membrane-bound proteins, we have demonstrated that protein-protein steric interactions can drive membrane curvature. These findings suggest that a simple imbalance in the concentration of membrane-bound proteins across a membrane surface can drive a membrane to bend, providing an efficient mechanism by which essentially any protein can contribute to shaping membranes.