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Elucidating the mechanics of clathrin-mediated endocytosis

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ABSTRACT

Clathrin-mediated endocytosis (CME) is a key metabolic pathway that plays a central role in the delivery of nutrients and drug carriers into cells. In this study, we model the interactions of lipid membranes with different types of protein scaffolds and active forces to provide mechanistic insights into CME. To this end, we develop and employ an extended theoretical framework of lipid membranes that entertains spatial heterogeneity and local anisotropy that could arise from membrane–protein interactions. We show that a departure from homogeneity and isotropy can lead to a variable surface tension field, conventionally assumed to be a constant parameter. We model the impact of resting tension in a cell and discuss its consequences on the minimal protein machinery needed to complete vesicle formation. Based on our quantitative model and findings, we highlight the physical principles that unify CME in apparently distinct yeast and mammalian cells.