BP 6: Membranes and Vesicles

Time: Thursday 13:30–14:45 Location: H6

Invited Talk BP 6.1 Thu 13:30 H6

How do lipids and proteins diffuse in cell membranes, and what do the diffusion experiments actually measure? — •Ilpo Vattulainen — Department of Physics, University of Helsinki

There are various techniques able to gauge diffusion in biomembranes. For instance, quasi-elastic neutron scattering measures diffusion in a non-perturbative manner over nanosecond time scales, yet sampling in space is in these experiments done over large distances. Meanwhile, single-particle tracking allows one to measure the dynamics of individual molecules in almost nanometer resolution, but these measurements are based on the use of markers that may interfere with the diffusion process. Here we discuss nanoscale simulation studies designed to explore the underlying molecular-scale diffusion mechanisms of lipids and membrane proteins. We also discuss the bases of single-particle tracking experiments by considering the effects of streptavidin-functionalized Au nanoparticle probes on lateral diffusion. The results show that lipids diffuse in a concerted fashion as clusters of lipids whose motion is highly correlated, and membrane proteins move as dynamical complexes with tens of lipids bound to the protein. Lipids linked to a streptavidin-nanoparticle complex also turn out to move in a concerted manner but as a complex with the linker protein and numerous non-labeled lipids, slowing down the motion of the probe by an order of magnitude. The results highlight that prior to using any technique, it is crucial to understand the physical basis of the diffusion process that one aims to measure. Otherwise, interpretation of experimental data can be a surprisingly difficult task.

BP 6.2 Thu 14:00 H6

Fusion of virus and host membranes - the role of virus geometry and matrix proteins — •Gonen Golani[1], Sophie Winter[2], Steffen Klein[2], Petr Chlanda[2], and Ulrich S. Schwarz[1] — [1]Institute for Theoretical Physics and BioQuant, Heidelberg University, D-69120 Heidelberg, Germany — [2]Schaller Research Groups, Department of Infectious Diseases-Virology, Heidelberg University Hospital, D-69120 Heidelberg, Germany

Many medically important viruses are enveloped by a lipid membrane, therefore, a crucial step in the infection process is the fusion of the viral and cellular membranes. The fusion pathway involves a series of non-bilayer intermediates configurations: First, the monolayers of the two opposing membranes merge to form a hemifusion connection, referred to as the stalk. Next, expansion of the stalk brings the distal lipid monolayers together into a hemifusion diaphragm. Lastly, opening and expansion of a fusion pore within the diaphragm completes the fusion process. The formation of the stalk and expansion of the fusion pore constitute the two major energy barriers in the process. While formation of the stalk is directly driven by the viral fusion proteins and was extensity studied in the last decades, pore expansion is less well understood. Here we compute the stresses in the diaphragm and the resulting energy barrier to fusion pore expansion. We analyze, for the first time, effect of the virus geometry and membrane-matrix interaction on viral fusion rate. We also suggest a model for the role of interferon-induced transmembrane proteins (IFITMs) in inhibition of fusion by increasing the energy barrier of fusion pore expansion.

BP 6.3 Thu 14:15 H6

Calponin-homology domain mediated bending of membrane associated actin filaments — Saravanan Palani[1,2], Sayantika Ghosh[1], Esther Ivorra-Molla[1], Scott Clarke[1], Andrejus Suchenko[1], Mohan Balasubramanian[1], and •Darius Köster[1] — [1]Centre for Mechanochemcial Cell Biology and Warwick Medical School, Division of Biomedical Sciences, CV4 7AL Coventy, UK — [2]Department of Biochemistry, Division of Biological Sciences, Indian Institute of Science, Bangalore-560012, India

Actin filaments are central to cell function and the actin cytoskeleton exhibits a variety of geometries. Here, we show that 'curly', the actin-binding calponin-homology domain and a C-terminal unstructured domain from the IQGAP family of proteins, stabilizes individual actin filaments in a highly curved geometry when anchored to lipid membranes. Whereas F-actin is semi-flexible with a persistence length of $10\mu m$, binding of mobile curly within lipid membranes generates actin filament arcs and full rings of high curvature with radii below $1\mu m$. Higher rates of fully formed actin rings are observed in the presence of the actin-binding coiled-coil protein tropomyosin and when actin is directly polymerized on lipid membranes decorated with curly. Strikingly, curly induced actin filament rings contract upon the addition of muscle myosin II filaments and expression of curly in mammalian cells leads to highly curved actin structures in the cytoskeleton. Taken together, our work identifies a new mechanism to generate highly curved actin filaments, which opens a range of possibilities to control actin filament geometries in vitro and in vivo.

BP 6.4 Thu 14:30 H6

Fission mechanisms of cylindrical membrane tubes — •Russell Spencer and Marcus Müller — Georg-August Universität Göttingen, Institute for Theoretical Physics, 37077 Göttingen, Germany

This work investigates the mechanisms and pathways for the fission of phospholipid membranes, in particular double-membrane fission as it occurs in mitochondrial division. We employ self-consistent field theory and utilize the string method to find the Minimum Free Energy Path (MFEP) connecting the metastable starting and ending states of different membrane topology in order to determine the most likely pathway for the transition. Our results suggest that the free energy barrier to membrane fission, as well as the dominant pathway, can be controlled by the tension experienced by the membrane. At high tension, the inner tube partially collapses into a worm-like micelle, which then ruptures, resulting in two capped tubes. The outer membrane then follows similarly. This pathway is non-leaky, i.e. the solvent inside the inner membrane, between the membranes and outside the outer membrane never mix. At lower tension, the barrier to forming a worm-like micelle becomes prohibitive, and instead, the inner and outer membranes fuse. This pathway is leaky as pores form close to the fusion sites.