Time: Monday 15:00-17:00

Location: H43

Topical Talk BP 5.1 Mon 15:00 H43 Cytoskeletal pattern formation: Self organization of driven filaments — • ANDREAS BAUSCH — Technische Universität München Living cells rely on the self organization mechanisms of cytoskeleton to adapt to their requirements. Especially in processes such as cell division, intracellular transport or cellular motility the controlled self assembly to well defined structures, which still allow a dynamic reorganization on different time scales are of outstanding importance. Thereby, the intricate interplay of cytoskeletal filaments, crosslinking proteins and molecular motors a central role. One important and promising strategy to identify the underlying governing principles is to quantify the physical process in model systems mimicking the functional units of living cells. Here I will present in vitro minimal model systems consisting of actin filaments, crosslinking molecules and myosin II exhibiting collective long range order and dynamics. I will discuss how a balance of local force exertion, alignment interactions, crosslinking and hydrodynamics affect the evolving dynamic structures.

BP 5.2 Mon 15:30 H43

Time dependent irreversible bundling of actin filaments with magnesium ions — TIMO MAIER^{1,2}, •TAMÁS HARASZTI^{1,2}, and JOACHIM P. SPATZ^{1,2} — ¹New Materials and Biosystems group, MPI for Intelligent Systems, Heisenbergstr. 3, 70569-Stuttgart, Germany — ²Biophysical Chemistry, University of Heidelberg, Im Neuenheimerg Feld 253, 69120-Heidelberg, Germany

Actin, an abundant protein in eukaryotic cells, forms filamentous structures playing critical roles in cellular adhesion, motility and determining the elastic properties and the shape of cells. Mixed with divalent cations, bundles were observed above a critical electrolyte concentration, e.g. $27 \, mM$ for Mg²⁺. The process is driven by counter ion condensation, but there are still unclear details. We have investigated two of these details using 2-dimensional networks of prepolymerized actin filaments, following the bundling process by the thermal motion of tracer particles attached to the bundles and fluorescence microscopy. Our results indicate, that the filaments preferentially adsorb the divalent magnesium ions, resulting in crosslinking down to $5 - 8 \, mM$ background concentration when the solution is provided by a mediate $(0.4 \,\mu l/min.$ loading rate) flow. While the driving forces are in the order of $0.1 - 0.25 \, pN$, as we have reported earlier in single actin experiments, the resulted bundling is not reversible by thermal motion even after removing the magnesium and adding EGTA to the solution for several hours.

BP 5.3 Mon 15:45 H43

The dynamical cytoskeleton regulates morphogenesis in rodlike bacteria — •Sven van Teeffelen — Department for Molecular Biology, Princeton University, Princeton, USA

Bacteria were long regarded as unstructured bags of freely diffusing proteins and DNA. Contrary to this view, bacterial cells are now known to display intricate sub-cellular localization and dynamics of their constituents, which is required for a variety of processes including cellular morphogenesis. Biophysically, one mechanism for achieving macroscopic order relies on the bacterial cytoskeleton. Here we report a quantitative study of the dynamics of the Escherichia coli actin homolog MreB, which is essential for the maintenance of rod-like cell shape in bacteria. We found that MreB rotates around the long axis of the cell in a persistent manner and that this rotation depends on the assembly of the peptidoglycan cell wall. Biophysical modeling suggests that MreB and cell-wall synthesis are physically coupled. Thus, the MreB motion observed constitutes a reporter of the local insertion of cell-wall material. In agreement with recent experiments on macroscopic twisting of the cell envelope during growth we find that peptidoglycan is deposited in the cell wall in a helical manner. The cell wall in turn ultimately determines bacterial cell shape. Semi-atomistic computational simulations suggest that one function of MreB is to ensure a uniform distribution of new peptidoglycan insertion sites, a necessary condition to maintain rod shape during growth. Based on the same computational framework we hypothesize that MreB governs bacterial cell shape in a non-trivial manner.

BP 5.4 Mon 16:00 H43 Super-resolution imaging of dynamic MreB filaments in B. **Subtilis - a multiple motor driven transport?** — PHILIPP VON OLSHAUSEN^{1,2} and •ALEXANDER ROHRBACH^{1,2} — ¹Lab for Bio- and Nano-Photonics, University of Freiburg, Georges-Koehler-Allee 102, 79110 Freiburg, Germany — ²Centre for Biological Signalling Studies (bioss), University of Freiburg

The cytoskeletal protein MreB is an essential component of the bacterial cell shape generation system. By a super-resolution variant of total internal reflection microscopy using structured illumination and by 3D stacks of deconvolved epi-fluorescence microscopy, we found that inside live Bacillus subtilis cells MreB forms filamentous structures of variable lengths, typically not longer than one micrometer. These filaments move mainly perpendicular to the long bacterial axis revealing a maximum velocity at an intermediate length and a decreasing velocity with increasing filament length. Filaments move along straight trajectories, but can reverse or alter their direction of propagation. Based on our measurements, we provide a model being able to explain all observations. In this model MreB filaments mechanically couple several motors that putatively synthesize the cell wall, whereas the filaments traces mirror the trajectories of the motors. Based on this idea, we developed a mathematical model that can explain the nonlinear velocity length dependence. We deduce that the coupling of cell wall synthesis motors determines the MreB filament transport velocity, whereas the filament mechanically controls a concerted synthesis of parallel peptidoglycan (PG) strands to improve cell wall stability.

BP 5.5 Mon 16:15 H43 Mechanochemical patterning of the PAR system in *C. elegans* — ●VIJAY KRISHNAMURTHY^{1,2}, JUSTIN BOIS³, FRANK JÜLICHER¹, and STEPHAN GRILL^{1,2} — ¹Max Planck Institute for the Physics of Complex Systems, Nöthnitzer Straße 38, 01187 Dresden, Germany. — ²Max Planck Institute for Molecular Cell Biology and Genetics, Pfotenhauer Straße 108, 01307 Dresden, Germany. — ³UCLA Department of Chemistry and Biochemistry, Los Angeles, CA 90095, USA.

The polarization of partitioning defective (PAR) proteins into anterior and posterior domains is a conserved mechanism in the zygotes of *Caenorhabditis elegans*. This segregation is driven by flows established in the actomyosin cortex. Passive advection of the PARs by these flows is known to lead a transient segregation of the PAR system. However, the mechanism by which the PAR proteins regulate the cortical flows is unclear. We present a model which incorporates the feedback of the PAR concentration fields into cortical flows, via a coupling to the local myosin concentration. This leads to stable segregated states of the PAR system. Our model provides a self-consistent and closed mechanism for the polarization of the PAR-actomyosin system which compares well with the known experimental facts.

BP 5.6 Mon 16:30 H43

Cell membrane deformation and its role during cytokinesis — •JOCHEN A. M. SCHNEIDER¹, ANDREA M. PEREIRA², EWA PALUCH², and GUILLAUME SALBREUX¹ — ¹Max Planck Institute for the Physics of Complex Systems, Dresden, Germany — ²Max Planck Institute of Cell Biology and Genetics, Dresden, Germany

Cytokinesis, the process of physically dividing the cell at the end of mitosis, is achieved through the regulated variations of forces within the cell. A key player in this process is the cell cortex, a thin layer of actin filaments and myosin molecular motors. Recently, Sedzinski *et al.* have introduced a mathematical model to describe the role of the cell cortex in cytokinesis. The model has shown that because of the contractile behavior of the cell cortex, the cell shape can be unstable with respect to symmetry breaking, and that cell elasticity is required for cell shape stability. We present here a potential role of the cell membrane in contributing to cell elasticity. The membrane is attached to the cell cortex and has to mechanically balance the difference between extracellular and intracellular pressure. Under simple hypothesis on the membrane mechanical behavior and its area regulation by the cell, we investigate how its interaction with the cortex influences cell shape.

BP 5.7 Mon 16:45 H43 Chromosome oscillations in mammalian cells — \bullet Federica TAVANO¹, NENAD PAVIN², and IVA M. TOLIC-NORRELYKKE¹ — ¹Max Planck Institute of Molecular Cell Biology and Genetics, 01307 Dresden, Germany — $^2 \mathrm{Department}$ of Physics, Faculty of Science, University of Zagreb, 10002 Zagreb, Croatia

Mitosis is the process by which eukaryotic cells replicate chromosomes into two identical sets that segregate to the nuclei of the two daughter cells. During mitosis, sister chromatids connect to mitotic spindle microtubules via protein complexes called kinetochores, and oscillate around the equatorial plane of the spindle. These oscillations are correlated with the microtubule plus-end dynamics. However, the mechanism of the oscillations is not known. Here we show that kinetochores in mammalian epithelial cells move roughly with a constant velocity until they switch the direction of movement. During the movement, the distance between sister kinetochores increases. During directional switches, sister kinetochores are not synchronized: The leading kinetochore stops while the trailing one continues moving, decreasing the distance between the sister kinetochores. These results suggest that the sister kinetochores are under tension during their movement, whereas they get compressed when they switch direction. Moreover, these data imply that the microtubules on the leading side undergo rescue before the microtubules on the trailing side undergo catastrophe. We propose that forces on the kinetochores synchronize microtubule dynamics, which is required for chromosome oscillations.