BP 23: Physics of Bacteria and Viruses

Time: Thursday 17:15–18:45 Location: ZEU 260

BP 23.1 Thu 17:15 ZEU 260

Membrane fluidity guides bacterial surface motility — •Claudia Holz[1], Berenike Maier[1], Jan Mehlich[2], and Bart Jan Ravoo[2] — [1]Universität Münster, Institut für allgemeine Zoologie und Genetik, Schlossplatz 5, 48149 Münster — [2]Universität Münster, Organisch-Chemisches Institut, Corrensstrasse 40, 48149 Münster

Bacterial surface motility enables bacteria to form microcolonies, colonise human host cells and abiotic surfaces, and is often required for biofilm formation. Twitching motility is powered by polymeric cell appendages called type IV pili. They act as grappling hooks that support motility by a cycle of pilus elongation, surface adhesion and retraction. It is very poorly understood how bacteria control the velocity and direction of twitching.

We investigated twitching motility of the human pathogen Neisseria gonorrhoeae on different surfaces including glass-supported membranes to mimic cell surfaces. We found that bacteria twitch with a velocity of ˜1μm/sec and that movement is persistent on a time scale of around 8sec. Velocity and persistence increased with decreasing fluidity of solid supported membranes. On micropatterned surfaces, bacterial movement was confined to the least fluid regions, i.e. we found that motility was guided by surface fluidity. Our experiments reveal an unprecedented physical mechanism for controlling the direction of twitching motility and we hypothesize that this mechanism is involved in formation of microcolonies during infection.

BP 23.2 Thu 17:30 ZEU 260

Min proteins in growing Escherichia coli: from stochastic switching to oscillations — •Elisabeth Fischer-Friedrich[1], Giovanni Meacci[2], Hugues Chate[3], and Karsten Kruse[4] — [1]Max Planck Institute for the Physics of Complex Systems, Nöthnitzer Straße 38, 01187 Dresden, Germany — [2]IBM T. J. Watson Research Center, P.O. Box 218, Yorktown Heights, NY 10598 — [3]CEA Service de Physique de l'Etat Condensé, 91191 Gif-sur-Yvette, France — [4]Theoretische Physik, Universität des Saarlandes, Postfach 151150, 66041 Saarbrücken, Germany

Self-organization of proteins in space and time is of crucial importance for the functioning of cellular processes. Often, this organization takes place in the presence of strong fluctuations due to the small numbers of proteins involved. We report on stochastic switching of the localization of Min proteins in short E. coli cells. In longer bacteria, the switching turns into regular oscillations that are required for positioning of the division plane. Considering the intrinsic fluctuations in a simple model reproduces stochastic switching as well as oscillatory behavior. This provides strong evidence for the fact that the macroscopic switching is rooted in the microscopic fluctuations of the molecular processes involved.

BP 23.3 Thu 17:45 ZEU 260

Spatio-Temporal Protein Dynamics in Single Bacteria Cells on Chip — •Dominik Greif[1], Nataliya Pobigaylo[2], Anke Becker[2], Jan Regtmeier[1], and Dario Anselmetti[1] — [1]Experimental Biophysics & Applied Nanoscience, Bielefeld University, Germany — [2]Genetics and Systems Biology of Prokaryotes, Albert-Ludwigs-University Freiburg, Germany

Single cell microscopy of bacterial cells is very challenging due to their small size that differs considerably from eukaryotic cells. In order to investigate subcellular processes in microorganisms we recorded high resolution time lapse fluorescence images (TLFI) for monitoring the dynamics of proteins.
Exemplarily, we present observations of the intracellular protein GcrA as well as the asymmetric localization of the protein DivK in daughter cells of *S.meliloti* over at least two cell divisions. GcrA is a master transcriptional regulator that activates expression of genes participating in DNA replication progression and chromosome partitioning. DivK is part of the phosphorelay system which causes up-regulated expression for proteins required in DNA methylation, septum formation, flagellar synthesis, and chemotaxis. Methodically, a high numerical aperture optical setup with a sensitive CCD camera and a poly(dimethysiloxane) (PDMS) microfluidic chip, assuring a very good cell viability, was used. For cell immobilization a concentration gradient of polyethyleneimine (PEI) was used. Our approach allows new insight in the control of the bacterial cell cycle in individual cells towards a better understanding of cell proliferation and differentiation.

BP 23.4 Thu 18:00 ZEU 260

In-vitro assembly of Polyoma VP1 — •Henning Seidel — Institute of Physics, Ratzeburger Allee 160, 23538 Lübeck, Germany

One essential element of a virus is its protein shell, the viral capsid, which encloses the viral genome. The murine Polyomavirus is a non-enveloped DNA tumor virus with an icosahedral T=7d structure. Besides the knowledge of the structure, it is of utter importance to understand the process of viral assembly. The assembly reaction of Polyoma VP1 does not show the typical sigmoidal kinetics in light scattering experiments. The apparent kinetics is of fourth order, which appears rather unrealistic. In order to gain knowledge of the capsid composition during assembly beyond ensemble average, we apply methods of single molecule fluorescence, namely fluorescence correlation spectroscopy (FCS), fluorescence-intensity-distribution-analysis (FIDA), and single-particle-imaging (SPI).

These will help to answer the main questions: Is there an initial phase to form a nucleus? Exist pronounced intermediates along the assembly pathway? After building the capsid, is there an exchange of pentameres between capsid (Breathing)?

BP 23.5 Thu 18:15 ZEU 260

Internal Capsid-Pressure Dependence of Viral Infection by Phage λ — •Sarah Köster[1,2], Alex Evilevitch[3], Meerim Jeembaeva[3], and David Weitz[2] — [1]Courant Research Centre Physics, University of Göttingen, Germany — [2]Department of Physics and School of Engineering and Applied Sciences, Harvard University, Cambridge, USA — [3]Department of Biochemistry, Lund University, Sweden

Ejection of the genome from the virus, phage λ, is the initial step in the infection of its host bacterium. In vitro, the ejection depends sensitively on internal pressure within the virus capsid; however, the effect of internal pressure on infection of bacteria is unknown. Here, we use microfluidic devices to produce monodisperse aqueous emulsion droplets in a continuous oil phase. The drops serve as individual, picoliter-sized compartments for cells and viruses and enable us to study organisms on the single cell level while providing valuable statistical information. We monitor individual cells and determine the temporal distribution of lysis due to infection as the capsid pressure is varied. The lysis probability decreases markedly with decreased capsid pressure. Interestingly, the average lysis times remain the same, but the distribution is broadened, as the pressure is lowered.

BP 23.6 Thu 18:30 ZEU 260

Mechanics of the influenza virus are dominated by its lipid bilayer — •Iwan A. T. Schaap, Frederic Eghiaian, John Skehel, and Claudia Veigel — National Institute for Medical Research, Mill Hill, London, UK

The influenza virus protects its RNA genome by a loosely ordered non-symmetric protein capsid that is enveloped by a lipid bilayer. This membrane contains the various proteins responsible for binding and fusion with the target cell. The composite architecture helps the virus to meet two apparently conflicting demands on its rigidity during its life cycle: 1) it should persist in the often hostile extracellular environment when it transfers from host to host, and 2) it should permit the viral membrane to fuse with the acidic compartments of the target cell to allow infection.

In order to understand how the various parts of the viral structure contribute to its mechanical properties we used atomic force microscopy and finite element modeling to characterize the mechanical performance of influenza virus and compared it to the response of several simplified model systems.

We have found that the elastic properties of the influenza virus are best described by membrane mechanics. In contrast to the symmetrical non-enveloped viruses, the relatively soft influenza virus does not rely on a though protein shell for its survival.