BP 45: Systems biology

Time: Thursday 15:00–16:15 Location: H 1028

BP 45.1 Thu 15:00 H 1028

Centriole Centering in Centrosomes: Behavior of Catalytic Particles in Active Droplets — •David Zwicker[1,2], Anthony A. Hyman[3], and Frank Jülicher[2] — [1]Harvard University, Cambridge MA, USA — [2]Max Planck Institute for the Physics of Complex Systems, Dresden, Germany — [3]Max Planck Institute of Cell Biology and Genetics, Dresden, Germany

Centrosomes are membrane-less organelles, which are important for cell division. A centrosome consist of liquid-like pericentriolar material that accumulates around a centriole pair. We describe the centrosome as an active droplet, where the pericentriolar material is created from soluble building blocks by chemical reactions that are catalyzed both inside the droplet and at the centrioles [D. Zwicker, M. Decker, S. Jaensch, A. A. Hyman, F. Jülicher, *PNAS* **111** E2636-45 (2014)]. This model accounts for the observed nucleation and growth behavior as well as the suppression of Ostwald ripening.

Here, we analyze this model further and focus on the effects of the material fluxes that are created by the interplay of chemical reactions and diffusion. In particular, we show that centrioles exhibit an effective centering force if their catalytic activity is large enough. Furthermore, fluctuations of the spherical droplet shape are suppressed, even for an arbitrarily small surface tension. The non-equilibrium conditions created by the chemical reactions thus allow to control important properties of active droplets.

BP 45.2 Thu 15:15 H 1028

Reaction-diffusion processes and molecular crowding — •David Gomez and Stefan Klumpp — Max Planck Institute of Colloids and Interfaces, Potsdam, Germany

The interior of a living cell is a highly crowded environment, with macromolecules occupying up to 20-40% of the total cell volume. Molecular crowding affects both the thermodynamics and the kinetics of molecular binding, via their equilibrium constant and the diffusion coefficients, respectively. Using random walk on a lattice as well as off-lattice simulations with the simulation software ReaDDy, we consider reactions that uncouple the effects of molecular crowding on binding equilibria and the effects on molecular diffusion. As an application of our method, we combine the two uncoupled reactions into a Michaelis-Menten-like reaction and study the effects of molecular crowding on the overall product synthesis rate. The conditions to obtain the maximal product synthesis rate depend on crowding levels and other parameters intrinsic to the reaction.

BP 45.3 Thu 15:30 H 1028

Spatio-temporal dynamics of segregation in gonococcal populations — •Enno R. Oldewurtel, Nadzeya Kouzel, and Berenike Maier — Department of Physics, University of Cologne

Various bacterial pathogens evolved to escape the host immune system by reversibly switching off the generation of surface molecules via mutations. This can generate heterogeneity within a population. However, new mutant cells are likely to get lost again due to stochastic fluctuations before increasing in number. Hence, it is unclear how heterogeneity can evolve and be maintained.

The human pathogen *Neisseria gonorrhoeae* can undergo frequent changes in its major virulence factor, a long polymeric cell appendage, called type IV pilus. It can switch on and off modifications or production of this structure. Type IV pili mediate aggregation among bacteria. Thus changes in the pilus, can lead to changes in the physicochemical interaction between cells. Here, we address the spatio-temporal dynamics of emergence and spreading of bacteria with modified or lacking type IV pili, within a growing colony of *N. gonorrhoeae*.

We are able to directly visualise mutants via fluorescent proteins. Mutants gaining the ability to modify their pili by glycosylation and mutants no longer producing pili, were seen to spread more easily within the population. We attribute this effect to decreased cell-to-cell interaction by either changing the pilus or lacking it.

We conclude that fine-tuning of physical interactions can lead to segregation into sub-populations, thus maintaining the heterogeneity and co-existence of multiple phenotypes.

BP 45.4 Thu 15:45 H 1028

Scaling and regeneration of self-organized patterns — •Steffen Werner[1], Tom Stückemann[2], Manuel Beirán Amigo[1,3], Jochen C. Rink[2], Frank Jülicher[1], and Benjamin M. Friedrich[1] — [1]Max Planck Institute for the Physics of Complex Systems, Dresden, Germany — [2]Max Planck Institute of Molecular Cell Biology and Genetics, Dresden, Germany — [3]Universidad Autónoma de Madrid, Madrid, Spain

Biological patterns and morphologies, generated during development and regeneration, often scale with organism size. Some organisms such as flatworms can even regenerate an appropriately scaled body plan from tissue fragments of varying sizes. Turing proposed a general principle for self-organized chemical pattern formation, yet the resulting Turing patterns usually do not scale with system size, but are governed by characteristic length scales. Here, we introduce a generalization of Turing patterns that is both self-organized and self-scaling. We analytically characterize this novel class of pattern forming systems, for which a Turing instability is coupled to the reaction kinetics of diffusing expander molecules. This expander regulates reaction rates of the Turing system, thereby adjusting its intrinsic length scale proportional to system size. Using dynamical systems theory, we identify minimal requirements for self-scaling. We address robustness of emerging patterns with respect to parameter variations as well as structural robustness of the feedback logic itself. Our model captures essential features of body plan regeneration in flatworm fragments as observed in amputation experiments. **For more information: arXiv:1411.2359**

BP 45.5 Thu 16:00 H 1028

Competition between nucleosomes and transcriptional machinery determines the timing of genome activation in the zebrafish embryo — •Stefanie Belohlavy[1], Jens Karschau[1], Shai R. Joseph[2], Mukesh Kumar[2], Andrej Shevchenko[2], Nadine L. Vastenhouw[2], and Vasily Zaburdaev[1] — [1]MPI PKS, Dresden, Germany — [2]MPI CBG, Dresden, Germany

In a developing embryo, many identical cells are formed by rapid cell divisions from one single egg cell. Initially, DNA is not readable for protein synthesis, and cells only rely on egg-provided resources. Then, once a certain number cells have been produced, DNA transcription starts. Why DNA is initially transcriptionally silent is a topic of strong debate. Previously, biological models of a repressor silencing DNA were proposed. In analogy to this idea, we experimentally show nucleosomes to act as a highly abundant repressor in zebrafish. To fully explain transcriptional activation, we propose a theoretical competition model. It consists of an activator (which only binds to specific sites) and a repressor (which has uniform probability to bind anywhere on the DNA). The exponential increase of DNA lowers the free repressor pool. Whenever a repressor leaves an activator site, activators rebind to it instead of a repressor triggering DNA transcription. Our model is consistent with our experimental advances: variations in the ratio of DNA to repressor shift the time of transcriptional onset. Future modifications to the strength of binding sites will help us to identify particular activators as theory and experiment continue to advance side by side.