# BP 16: Systems Biophysics

Time: Wednesday 11:15–13:00 Location: BAR 0106

**Invited Talk** BP 16.1 Wed 11:15 BAR 0106

**Systems biophysics of bacterial response to cell wall-targeting antibiotics** — Rebecca Brouwers[1], Sharareh Tavaddod[1], Leonardo Mancini[2], Jacob Biboy[3], Elizabeth Tatham[1], Pietro Cicuta[2], Waldemar Vollmer[3], and •Rosalind Allen[1,4] — [1]School of Physics & Astronomy, University of Edinburgh, Edinburgh, UK — [2]Cavendish Laboratory, University of Cambridge, Cambridge, UK — [3]Centre for Bacterial Cell Biology, University of Newcastle, Newcastle, UK — [4]Theoretical Microbial Ecology, Faculty of Biological Sciences, University of Jena, Germany

Antibiotics are central in modern medicine, yet bacterial infections are increasingly becoming resistant to antibiotics. To use antibiotics more effectively, we need to understand better how they work. We have used a combination of microbiological and biophysical experiments, and theoretical modelling, to probe how the antibiotic mecillinam, which targets bacterial cell wall synthesis, kills the bacterium Escherichia coli. Comparing killing dynamics under conditions of rich and poor nutrients we conclude that the balance between the rates of creation of cell surface area and volume plays a crucial role in the fate of cells when exposed to this antibiotic.

BP 16.2 Wed 11:45 BAR 0106

**Cell size distribution: an analytical comparison between lineage and population experiments** — •Arthur Genthon — ESPCI, Université PSL, Paris, France (until december 2022) — Max Planck Institute for the Physics of Complex Systems, Dresden, Germany (from january 2023)

In the past decade, new microfluidic devices, like the mother machine, have been designed to monitor single lineages of cells for many generations with great precision. In classical bulk cultures where full populations are grown, cells with high reproductive success lead to larger populations of offsprings, while no such selection effect is present in single-lineage experiments. Quantifying the statistical bias between these two families of experiments is at the basis of a universal notion of natural selection, which can be defined for any branching tree, not just in cell biology. In this work, we thus compute analytical lineage-population biases for the cell size distribution, in the context of size-controlled cells. The role of stochasticity, both in single-cell growth and in volume partitioning at division, is explored, and we show how it can cancel the lineage-population bias. In addition, in simple cases we show how we can learn the laws of cell growth and division from mother machine steady state size distributions. The parameters of the model, such as the single-cell growth rate, the strength of the size control or the asymmetry of division are obtained by fitting analytical distributions to Escherichia coli data.

BP 16.3 Wed 12:00 BAR 0106

**Tuning pattern formation of *E. coli* Min proteins *in vivo*** — Ziyuan Ren[1], •Henrik Weyer[2], Laeschkir Würthner[2], Erwin Frey[2], and Suckjoon Jun[1] — [1]Department of Physics & Section of Molecular Biology, Division of Biological Sciences, University of California San Diego, 9500 Gilman Dr. La Jolla, CA 92093, USA — [2]Arnold Sommerfeld Center for Theoretical Physics and Center for NanoScience, Department of Physics, Ludwig-Maximilians-Universität München, Theresienstraße 37, D-80333 München, Germany

The Min protein system of *Escherichia coli* bacteria is crucial for their proliferation. The Min proteins ensure symmetric cell division by positioning the cell-division machinery at midcell. This spatial templating is achieved by the self-organized pole-to-pole oscillation of the Min proteins, suppressing FtsZ ring formation at the cell poles. We experimentally study the robustness of Min pattern formation under changes in the total protein content of MinD and MinE by genetically modifying their expression in live *E. coli* bacteria. This uncovers a remarkable robustness of Min patterns *in vivo* comparable with previous findings *in vitro*. Moreover, this study reveals that the protein concentrations determine the pattern type, and both standing-wave and traveling-wave patterns form in filamentous cells. We show that the same reaction–diffusion model based on the conformational switch of MinE introduced earlier for the *in vitro* system explains both the robustness and the pattern characteristics *in vivo*. Thus, common principles underlie Min pattern formation *in vivo* and *in vitro*.

BP 16.4 Wed 12:15 BAR 0106

**Stochastic dynamics of cell shape during cellular state transitions** — •Wolfram Pönisch[1], Iskra Yanakieva[1], Aki Stubb[2], Guillaume Salbreux[3], and Ewa Paluch[1] — [1]Dept. of Physiology, Development and Neuroscience, University of Cambridge, Cambridge, UK — [2]Stem Cells and Metabolism Research Program, University of Helsinki, Helsinki, Finland — [3]Dept. of Genetics and Evolution, University of Geneva, Geneva, Switzerland

The development of an organism is characterized by a series of cellular state transitions where cells become increasingly specialized. Such state transitions are often accompanied by morphological changes and there are strong indications of coupling between a cell's shape and state. Here, we present a pipeline to quantify and analyse cell shapes as cells undergo the epithelial-to-mesenchymal transition (EMT). We apply our analysis pipeline to study how shape and fate are coupled during the EMT of MDCK cells. We confirm that cell morphology is closely associated with the state: While epithelial cells display spherical shapes, mesenchymal cells undergo spreading. After defining the distinct cellular shapes corresponding to cell states, we study how exactly the morphological features of a cell evolve during EMT. To this aim, we investigate trajectories of the morphological features of individual cells in a low-dimensional morphospace and describe the evolution of cellular shape as a Langevin process, allowing us to entangle the role of deterministic and stochastic morphogenetic forces. By integrating morphometric analysis into studies of cell state transitions, we aim to better understand the crosstalk between cell state and shape.

BP 16.5 Wed 12:30 BAR 0106

**Sensitivity of Boolean attractors to small network changes and implications for the inference of microbial interaction networks** — •Jyoti Jyoti and Marc-Thorsten Hütt — Jacobs university, Bremen, Germany

Sensitivity of dynamics on graphs under small topological changes has been studied for diverse types of dynamics and in relation with a wide range of applications. Here we extend this direction of research by studying (and deciphering) the change of attractors in Boolean threshold dynamics under single and multiple edge switches. By evaluating differences in attractor sets, we can, with high accuracy, predict the structural change in the network. This is of high relevance for network inference, e.g., inferring microbial interactions from abundance patterns: Current approaches, where interaction networks are inferred from attractors [1], often fail to provide networks, which in turn can reproduce the initial attractor set. Evaluating the differences in attractor sets (initial set vs. the one produced by the inferred network) and estimating topological differences from them can pave the way towards better inference algorithms. We briefly discuss the implications of our findings for microbiome analyses.

[1] Claussen, J. C., Skiecevičienė, J., Wang, J., Rausch, P., Karlsen, T. H., Lieb, W., Baines, J. F., Franke, A., and Hütt, M.-Th. (2017). Boolean analysis reveals systematic interactions among low-abundance species in the human gut microbiome. *PLoS Computational Biology*, 13(6):e1005361.

BP 16.6 Wed 12:45 BAR 0106

**Information storage allows for optimal adaptation in chemical signalling networks out-of-equilibrium** — •Daniel Maria Busiello[1] and Giorgio Nicoletti[2] — [1]Max Planck Institute for the Physics of Complex Systems, Germany — [2]University of Padua, Italy

Living systems process information and exhibit dynamical adaptation. We propose a chemical model for sensing that encompasses only necessary ingredients: energy consumption, information storage, and negative feedback. Indeed, equilibrium constraints limit the efficiency of information processing, and storage is an unavoidable energy-consuming step to exploit information. Our model architecture is informed by experimental observations that found negative feedback to be ubiquitous. We show that the presence of information storage and negative feedback leads to finite-time memory, essential for dynamical adaptation. Surprisingly, adaptation is associated with both an increase in the mutual information between external and internal variables and a reduction of dissipation in the internal chemical processes. This twofold advantage comes at an energetic cost. By simultaneously optimising energy consumption and information processing features, we find that

far-from-equilibrium sensing dominates in the low-noise regime. Finally, we employ our model to shed light on the adaptation of neurons in zebrafish larvae subjected to periodic visual stimuli. We find striking similarities between predicted and observed behaviours, quantifying dissipation and information-processing performance. Our theory provides a stepping stone towards the idea of highlighting crucial ingredients for information processing starting from a chemical description.