BP 17: Multicellular Systems II

Time: Tuesday 11:00–13:30 Location: BPb

BP 17.1 Tue 11:00 BPb

Encoding memory in biological network hierarchy — •Mirna Kramar[1] and Karen Alim[1,2] — [1]Max Planck Institute for Dynamics and Self-Organization, 37077 Göttingen, Germany — [2]Physik-Department, Technische Universität München, Garching, Germany

Remembering sources of food and threat is essential for survival. Even very simple organisms are able to encode sensory information that aids them in tackling complex environments. The slime mould *Physarum polycephalum* is a giant unicellular eukaryote whose body consists of a network of tubes which undergoes constant reorganization. The mechanism behind the network reorganization upon food encounter has not been explained previously. Here, we identify the imprint the food stimulus leaves on network morphology as memory and show that the network relies on tube growth and flows to encode stimulus information. We hypothesise an encoding mechanism introducing a local release of a chemical agent that affects the mechanical properties of the tubes and spreading through the network by protoplasmic flows. Using a theoretical model, we test our hypothesis and find the model yields a correct prediction of flow-dependent stimulus response. Finally, we investigate the role of network hierarchy in memory encoding and show that the network directly relies on existing tube diameter hierarchy to encode the stimulus. Our findings [1] demonstrate *P.polycephalum*'s ability to encode and read stored memory and likely open doors to the use of the organism in bioinspired design.

[1] Kramar and Alim, PNAS, in press (2021)

BP 17.2 Tue 11:20 BPb

A lumped-parameter model illustrates information processing and migration in the slime mold *Physarum polycephalum* — •Christina Oettmeier and Hans-Günther Döbereiner — Institut für Biophysik, Universität Bremen

The slime mold *P. polycephalum* exhibits rich spatiotemporal oscillatory behavior. The organism's size spans orders of magnitude, from large meter-sized stationary transport networks down to micrometer-sized amoebae. All morphotypes show actomyosin-based contraction-relaxation cycles resulting in protoplasmic streaming. Furthermore, the giant amoeba shows a very high behavioral plasticity, leading to speculations about the origins of cellular minimal cognition. The underlying functions are not neuron-based, but are emergent phenomena, resulting from mechanochemical processes on the tubular network. In this context, we investigate how the slime mold processes information. At different parts of a migrating amoeba, oscillation frequencies vary. Oscillations in the back cause endoplasm flows through the internal vein system and expand the frontal membrane. We use the electronic-hydraulic analogy, implemented in a lumped-parameter model, to investigate this special case of information processing. A single vein segment can be described as a flexible tube, possessing a fluidic resistance (R) and fluidic capacitance (C) due to wall elasticity. The electronic equivalent is a passive RC low pass filter. Thus, the oscillation frequencies at the back are higher than those at the front due to filtering. The model can also explain the onset of locomotion.

BP 17.3 Tue 11:40 BPb

Morphoelasticity of Large Bending Deformations of Cell Sheets during Development — •Pierre A. Haas[1,2,3] and Raymond E. Goldstein[4] — [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]Center for Systems Biology, Dresden, Germany — [4]Department of Applied Mathematics and Theoretical Physics, University of Cambridge, United Kingdom

Deformations of cell sheets during morphogenesis are driven by developmental processes such as cell division and cell shape changes. In elastic shell theories of development, these processes appear as variations of the intrinsic geometry of a thin shell. However, morphogenesis often involves large bending deformations that are outside the formal range of validity of classical shell theories.

In this talk, I will therefore discuss a shell theory valid in this new geometric limit of large bending deformations [1]. I will emphasise the geometric material anisotropy that arises in this theory and the elastic role of cell constriction. Finally, taking the invagination of the spherical embryos of the alga *Volvox* as a model, I will compare this shell theory to a classical theory not formally valid for large bending deformations and reveal how the geometry of large bending deformations stabilises invagination [1].

[1] P. A. Haas and R. E. Goldstein, Phys. Rev. E **103** (2021, in press)

BP 17.4 Tue 12:00 BPb

Migration of immune cells in an obstacle park — •Doriane Vesperini[1], Zeinab Sadjadi[2], Heiko Rieger[2], and Franziska Lautenschläger[1] — [1]Experimental Physics, Saarland University, 66123 Saarbrücken, Germany — [2]Theoretical Physics, Saarland University, 66123 Saarbrücken, Germany

Several crucial processes in biological systems can be described as a search problem such as: finding food resources or pathogens. The presence of obstacles like non-targeted cells or extracellular matrix in biological environments induces a perturbation of the initial cell trajectory. For example, the presence of bystander cells has been shown to increase the velocity and the persistence of natural killer cells [1]. Besides obstacles density, their spatial disposition may also influence the search efficiency. It has been demonstrated that the density and geometry of pillar lattices affect migration strategies of cells [2].

We investigate how search efficiency is influenced by spatial arrangement of obstacles. A microfluidic device is designed to track HL60 cells differentiated into neutrophils in confined 2D environments. Our device consists of pillar forests distributed in triangular or square arrangements. We calculate the escape time and diffusion properties of the searcher in different densities and height of pillars and investigate which key parameters influence the search efficiency.

[1] Zhou X., et al. Scientific Reports (2017)
[2] Gorelashvili M., et al. New Journal of Physics (2014)

BP 17.5 Tue 12:20 BPb

Cell-cell adhesion and 3D matrix confinement explain plasticity of breast cancer invasion — •Simon Syga[1], Peter Friedl[2,3,4], and Andreas Deutsch[1] — [1]Center for Information Services and High Performance Computing, Technische Universität Dresden, Germany — [2]Department of Cell Biology, Radboud Institute for Molecular Life Sciences, Radboud University Medical Center, Nijmegen, the Netherlands — [3]David H. Koch Center for Applied Genitourinary Cancers, The University of Texas MD Anderson Cancer Center, Houston, TX, USA — [4]Cancer Genomics Centre, Utrecht, the Netherlands

Plasticity of cancer invasion and metastasis depends on the ability of cancer cells to switch between collective invasion modes and single cell dissemination, under the control of cadherin-mediated cell-cell junctions. E-cadherin is considered a tumor suppressor, the downregulation of which causes single-cell scattering in 2D environments. In clinical samples, however, E-cadherin expressing and deficient tumors both invade collectively and metastasize equally, implicating additional mechanisms controlling cell-cell cooperation and dissemination. Using a cellular automaton model we identify physical confinement by the extracellular matrix (ECM) as the dominant physical mechanism that supports collective invasion irrespective of the composition and stability of cell-cell junctions. In particular, we predict that downregulation of E-cadherin results in a transition from coordinated to uncoordinated collective movement along extracellular boundaries, whereas single-cell escape depends on locally free tissue space.

BP 17.6 Tue 12:40 BPb

Learning the dynamics of cell-cell interactions in confined cell migration — •David Brückner[1], Nicolas Arlt[2], Alexandra Fink[1], Pierre Ronceray[3], Joachim Rädler[2], and Chase Broedersz[1,4] — [1]Arnold Sommerfeld Center for Theoretical Physics and Center for NanoScience, Ludwig-Maximilians-Universität, München — [2]Faculty of Physics and Center for NanoScience, Ludwig-Maximilians-Universität, München — [3]Center for the Physics of Biological Function, Princeton University, Princeton, NJ 08544, USA — [4]Department of Physics and Astronomy, Vrije Universiteit Amsterdam, 1081 HV Amsterdam, The Netherlands

Contact-mediated cell-cell interactions play a key role in shaping the stochastic trajectories of migrating cells. But how can we describe the stochastic dynamics of colliding cells in a unifying theoretical frame-

work? To address this question, we monitor stochastic cell trajectories in a micropatterned cell collider in which pairs of cells perform repeated cellular collisions. Capitalizing on this large experimental data set of coupled cell trajectories, we infer an interacting stochastic equation of motion that accurately predicts the observed interaction behaviors. Our approach reveals that interacting non-cancerous MCF10A cells can be described by repulsion and friction interactions. In contrast, cancerous MDA-MB-231 cells exhibit novel and surprising attraction and anti-friction interactions, promoting the predominant relative sliding behavior observed for these cells. Based on the inferred interactions, we show how our framework may generalize to provide a unifying theoretical description of diverse cellular interaction behaviors.

30 min. Meet the Speaker