BP 7: Cell Mechanics, Cell Adhesion and Migration, Multicellular Systems

Time: Thursday 15:00–16:15 Location: H6

Invited Talk BP 7.1 Thu 15:00 H6

Shaping embryos through controlled tissue phase transitions — •Otger Campàs — Physics of Life Excellence Cluster, TU Dresden, Germany — University of California, Santa Barbara, USA

During embryonic development, cells self-organize to build functional structures, like tissues and organs, and progressively shape the organism. While many key molecular players that orchestrate embryonic development are known, the physical mechanisms underlying embryonic morphogenesis remain largely unknown, mainly because of a lack in methodologies enabling direct in vivo and in situ measurements of forces and mechanical properties within developing 3D tissues and organs. For similar reasons, understanding the fundamental physical nature of active multicellular systems has been very challenging. We have recently developed novel microdroplet-based techniques that allow direct quantitative measurements of mechanical forces and material properties within 3D multicellular systems, including developing embryonic tissues. Using these techniques and focusing on the elongation of the body axis, a hallmark morphogenetic process in vertebrate development, we reveal a new physical mechanism of tissue morphogenesis whereby spatiotemporally controlled fluid-to-solid (rigidity) transitions in the tissue physical state, rather than patterned mechanical stresses, guide tissue flows to shape functional embryonic structures. Moreover, combining computational and experimental data, we show that active tension fluctuations control tissue fluidization in vivo.

BP 7.2 Thu 15:30 H6

Traction force microscopy with invertible neural networks — •Johannes Blumberg[1], Timothy Herbst[1,2], Ullrich Koethe[2], and Ulrich Schwarz[1] — [1]Institute for Theoretical Physics and Bioquant, Heidelberg University — [2]Visual Learning Lab, IWR, Heidelberg University

In traction force microscopy (TFM), the mechanical forces of cells adhering to an elastic substrate are estimated from the substrate displacements as measured by the movement of embedded fiducial marker beads. While the direct problem of calculating displacement from forces is well-defined by elasticity theory, the inverse problem of reconstructing forces from displacements is ill-posed. Usually an estimate is obtained by minimizing the mean squared distance between experimentally observed and predicted displacements. The standard method in this regard is Fourier Transform Traction Cytometry (FTTC), whose superior efficiency is based on the convolution theorem in Fourier space. Here we explore if the performance can be improved by using machine learning methods, in particular invertible neural networks, which recently have emerged as powerful method to solve ill-posed inverse problems.

BP 7.3 Thu 15:45 H6

An active gel model for optogenetic control of cell migration — •Oliver M. Drozdowski[1,2], Falko Ziebert[1,2], and Ulrich S. Schwarz[1,2] — [1]Institute for Theoretical Physics, Heidelberg University, Philosophenweg 19, 69120 Heidelberg, Germany — [2]BioQuant, Heidelberg University, Im Neuenheimer Feld 267, 69120 Heidelberg, Germany

Optogenetics has emerged as a new powerful experimental method to control cellular processes in space and time, including actin filament polymerization and contractility of myosin II molecular motors. Here we report on a mathematical analysis of spatiotemporal activation patterns in a simple one-dimensional variant of active gel theory with the aim to predict how optogenetics can be used to control cell migration [1]. We first show that the model can describe the symmetrical flow of the actomyosin system observed in optogenetic experiments but not the long-lasting polarization required for cell migration. Motile solutions, however, become possible if cytoskeletal polymerization is included through the boundary conditions. Optogenetic activation of contraction can then initiate locomotion in a symmetrically spreading cell and strengthen motility in an asymmetrically polymerizing one. If designed appropriately, it can also arrest motility even for protrusive boundaries.

[1] https://arxiv.org/abs/2104.14636, to appear in Phys. Rev. E

BP 7.4 Thu 16:00 H6

Defect-mediated morphogenesis — •Ludwig A. Hoffmann, Livio N. Carenza, Julia Eckert, and Luca Giomi — Universiteit Leiden, The Netherlands

Growing experimental evidence indicates that topological defects could serve as organizing centers in the morphogenesis of tissues. We provide a quantitative explanation for this phenomenon, rooted in the buckling theory of deformable active polar liquid crystals. Using a combination of linear stability analysis and computational fluid dynamics, we demonstrate that confined cell layers are unstable to the formation of protrusions in the presence of disclinations. The instability originates from an interplay between the focusing of the elastic forces, mediated by defects, and the renormalization of the system's surface tension by the active flow. The post-transitional regime is also characterized by several complex morphodynamical processes, such as oscillatory deformations, droplet nucleation and active turbulence. Our findings offer an explanation of recent observations on tissue morphogenesis and shed light on the dynamics of active surfaces in general.