DY 17: Focus Session: Nonlinear Dynamics in Biological Systems II (joint session DY/BP)

Nonlinear dynamics play a central role for biological systems to achieve remarkable complexity and adaptability. They underlie processes where small changes cascade into large effects, critical thresholds drive transitions, and feedback mechanisms maintain intricate balances. Biological systems are often far from equilibrium, exhibiting behaviors shaped by competing forces, stochastic fluctuations and emergent behavior. From the amplification of sensory signals near bifurcation points to the development of turbulence, concepts from nonlinear dynamics provide a unifying framework for studying patterns, stability, and collective behavior in living systems. This focus session explores the richness of nonlinear dynamics across biological scales, from molecular circuits to population-level phenomena, spanning vastly different fields from cardiac dynamics, embryogenesis and cell motility to active fluids, condensates and origin of life. Through theoretical models, experimental insights, and computational approaches, the talks illustrate how nonlinear-dynamics principles unravel the mechanisms driving function and complexity in biology, offering new perspectives across disciplines.

Organized by Philip Bittihn (Göttingen), Stefan Klumpp (Göttingen), and Carsten Beta (Potsdam)

Time: Tuesday 14:00–15:15 Location: H43

Invited Talk DY 17.1 Tue 14:00 H43

Mechanistic origins of temperature scaling in the early embryonic cell cycle — •Lendert Gelens — Laboratory of Dynamics in Biological Systems, Department of Cellular and Molecular Medicine, KU Leuven, Herestraat, 49, Leuven, Belgium

Temperature profoundly impacts organismal physiology and ecological dynamics, particularly affecting ectothermic species and making them especially vulnerable to climate shifts. Even though complex physiological processes usually involve dozens of enzymes, empirically it is found that the rates of these processes often obey the Arrhenius equation, which was originally derived for single enzyme-catalyzed reactions. Here we have examined the temperature scaling of the early embryonic cell cycle, with the goal of understanding why the Arrhenius equation approximately holds, and why it breaks down at temperature extremes.

Using experimental data from different frog, fish, fly, and worm species, we find that the apparent activation energies for the early embryonic cell cycle for diverse ectotherms are all similar. Computational modeling and experiments with frog egg extracts show that the non-Arrhenius scaling can be accounted for by biphasic temperature scaling in critical individual components of the cell cycle oscillator circuit, in combination with imbalances in the activation energies for different partially rate-determining enzymes. These findings provide mechanistic insights into the dynamic interplay between temperature and complex biochemical processes, and into why biological systems fail at extreme temperatures.

DY 17.2 Tue 14:30 H43

Reshaping morphogen gradients through porous tissue architecture — •Diana Khoromskaia[1,2] and Zena Hadjivasiliou[1,2,3] — [1]Francis Crick Institute, London, United Kingdom — [2]University College London, London, United Kingdom — [3]London Centre for Nanotechnology, London, United Kingdom

The morphogenesis of tissues during embryonic development is controlled by concentration gradients of morphogens – signalling molecules whose readout determines cell fate decisions. How the spread of morphogens is affected in tissues with complex geometry and spatially heterogeneous architecture is not well understood. To address this question, we introduce a porous vertex model, by explicitly considering the network of extracellular spaces between the cells. Morphogens produced by source cells disperse through the tissue via three modes of transport: extracellular diffusion, membrane-bound diffusion, and cell-based transport through recycling. With this model we investigate numerically and analytically how cell-scale geometry, such as cell size, cell shape anisotropy, and cell distance, influences effective diffusion and degradation of morphogens at tissue-scale. We further show that a non-linear coupling between cell packing and morphogen concentration renders the morphogen gradient robust to perturbations, for instance by locally buffering fluctuations in the production. Our characterisation of tissues as active porous materials provides new insights into how morphogenesis and cell fate determination may interact during embryonic development.

DY 17.3 Tue 14:45 H43

Active viscoelastic condensates provide controllable mechanical anchor points — •Oliver Paulin[1], Luise Zieger[2,3], Júlia Garcia-Baucells[5], Alexander Dammermann[5], Sebastian Aland[2,3,4], and David Zwicker[1] — [1]Max Planck Institute for Dynamics and Self-Organization, Göttingen — [2]TU Bergakademie Freiberg — [3]HTW Dresden — [4]Center for Systems Biology, Dresden — [5]Max Perutz Labs, University of Vienna

Many biological materials must couple mechanical strength with the ability to rapidly self-assemble at a specific location. In particular, biomolecular condensates readily self-assemble via phase separation, but may also need to anchor external forces to fulfil their function. Spatial localisation of condensate formation can be controlled by active cores that preferentially drive the production of condensate material at a particular point, while resistance to external forces can be facilitated by viscoelastic material properties. Here, we develop a continuum model of viscoelastic growth around an active core, and investigate the results in a spherically symmetric geometry. We find that viscoelastic stresses restrict condensate growth, but also impart resistance to deformation. We investigate the effect of varying different mechanical properties on condensate growth and strength, and also study how strain-dependent material incorporation may limit the maximum rate of growth. Finally, we compare the predictions of our model to experimental data from centrosomes in C. elegans embryos, identifying a parameter regime in which rapid growth can be combined with appropriate mechanical strength.

DY 17.4 Tue 15:00 H43

Modelling cell crawling on different substrate stiffness — Sohei Nakamura and •Mitsusuke Tarama — Kyushu University, Fukuoka, Japan

Crawling cells sense the mechanical properties of the underlying substrate and change their dynamics accordingly. This ability called durotaxis is of great importance in various biological processes including development and homeostasis. In order to understand how intracellular chemical reactions and cellular mechanics give rise to durotaxis, we constructed a simple model from reaction diffusion equations for intracellular chemical compounds and force balance equations for the intracellular mechanics including the effect of the substrate stiffness. We found that within the model, the cell speed and diffusion coefficient change non-monotonically with the substrate stiffness, indicating the existence of an optimal substrate stiffness for migration. This non-monotonic behavior of the cell speed is consistent with experimental observations and can be understood to be caused by the competition between substrate adhesion and cell shape deformation. We further discuss cell migration on a patterned substrate.