BP 6: Statistical Physics of Biological Systems 1 (joint session BP/DY)

Time: Monday 15:00–17:15 Location: H16

BP 6.1 Mon 15:00 H16

Dynamics and Fair Risk Sharing in Groups of Intelligent, Egoistic Individuals — Samuel Monter[1], •Veit-Lorenz Heuthe[1], Emanuele Panizon[2], and Clemens Bechinger[1] — [1]FB Physik, Universität Konstanz, Konstanz, Germany — [2]Department of Quantitative Life Science, ICTP, Trieste, Italy

Many animal species organize in social groups of fascinating complexity. The evolutionary biologist W.D. Hamilton hypothesized that the gregariousness of some animals can be explained solely from the egoistic motivation to decrease the risk of predation [1]. As a quantitative measure of this risk, he considered the Voronoi area around each animal. Many collective behavior studies try to capture this motivation by imposing interaction rules or neglect the driving motive altogether when modeling the dynamics of animals. In this study we train a swarm of individuals in a Multi Agent Reinforcement Learning (MARL) framework according to Hamilton's hypothesis, i.e. to decrease their predation risk. Thus, we gain insights into the dynamics of an ensemble of selfishly motivated individuals unbiased by any a priori assumption about interactions. We find that the individuals learn to cluster into groups which exhibit dynamic steady states resembling the behavior of natural swarms. Additionally, the predation risk is shared evenly within the groups, counterintuitive to the selfish motivation of each individual. Our findings suggest that gregariousness could indeed be driven by selfish motives in accordance with Hamilton's hypothesis.

[1] W. D. Hamilton, Journal of theoretical Biology 1971, 31, 295-311.

BP 6.2 Mon 15:15 H16

Boundary-driven epithelial ordering: from the mouse embryo to topological defects — •Pamela Guruciaga[1], Takafumi Ichikawa[2], Takashi Hiiragi[3], and Anna Erzberger[1] — [1]European Molecular Biology Laboratory, Heidelberg, Germany — [2]Kyoto University, Kyoto, Japan — [3]Hubrecht Institute, Utrecht, The Netherlands

In physical problems boundaries are typically considered to be simple, static and externally fixed. Biological systems however not only interact with their surroundings, but also alter them in ways that feed back on their own dynamics. We address this complex interaction in the context of epithelial development. Motivated by observations of an interplay between apico-basal polarity and boundary geometry in mouse epiblast morphogenesis, we develop a theory for epithelial ordering based on the Landau-de Gennes approach to surface-induced order in liquid crystals. We introduce a vector order parameter to represent the polarity, and model its interaction with the boundaries by a weak anchoring energy. We calculate the alignment fields arising from different boundary curvatures, and compare our predictions with imaging data of the morphogenetic process. Our work highlights the role of extraembryonic tissue in embryogenesis, while identifying interesting physical phenomena, such as boundary-dependent transitions in the structure of topological defects.

BP 6.3 Mon 15:30 H16

A competitive advantage through fast dead matter elimination in confined cellular aggregates — •Yoav G. Pollack[1,2], Philip Bittihn[1], and Ramin Golestanian[1,3] — [1]Max Planck Institute for Dynamics and Self-Organization (MPI-DS), Göttingen, 37077, Germany. — [2]Max Planck Institute for Multidisciplinary Sciences (MPI-NAT), Göttingen, 37077, Germany. — [3]Rudolf Peierls Centre for Theoretical Physics, University of Oxford, Oxford, OX1 3PU, UK.

Competition of different cell types for limited space is relevant in biological processes such as tissue morphogenesis and tumor growth. Predicting the outcome for non-adversarial competition of such growing active matter is non-trivial, as it depends on how processes like growth, proliferation and the degradation of cellular matter are regulated in confinement; regulation that happens even in the absence of competition to achieve homeostasis. We show that passive by-products of the processes maintaining homeostasis can significantly alter fitness, enabling cell types with lower homeostatic pressure to outcompete those with higher homeostatic pressure. We reveal that interfaces play a critical role for this specific kind of competition: There, growing matter with a higher proportion of active cells can better exploit local growth opportunities that continuously arise as the active processes keep the system out of mechanical equilibrium. Our results show that optimizing the ratio of growing (active) to dead (passive) cells can be as important to survival as growth rates and their sensitivity to mechanical cues.

BP 6.4 Mon 15:45 H16

A biophysical model of DNA methylation ageing — •Aida Hashtroud and Steffen Rulands — Max Planck Institute for the Physics of Complex Systems, Dresden, Germany

Machine learning models can accurately predict biological age and time of death based on sequencing measurements of DNA methylation marks. The mechanistic basis underlying these methylation clocks is poorly understood. Here, using a combination of tools from statistical physics and sequencing experiments we show that biological age can be predicted as a result of collective processes in the boundaries between genomic regions of different densities of cytosine-guanine pairs (CpGs). Specifically, we define a biophysical model predicting the time evolution of DNA methylation patterns during ageing based on a wave localization mechanism of tilted competition between antagonistic chromatin modifiers. Our work shows that biological age can be predicted from DNA methylation patterns using models with few parameters inspired by statistical physics.

15 min. break

Invited Talk BP 6.5 Mon 16:15 H16

From active bacterial microcolonies to biofilms as model tissues — •Vasily Zaburdaev — Friedrich-Alexander-Universität Erlangen-Nürnberg, Erlangen, Germany — Max-Planck-Zentrum für Physik und Medizin, Erlangen, Germany

Bacterial intrinsic activity is evident on all stages of their life cycle. We will start by following how individual cells deploy forces to attach and move on surfaces. We suggest how these active movements may be harnessed to generate work and, for example, cells can power the rotation of micro-turbines. When let to move and interact, however, bacteria will find each other and form microcolonies that consist of several thousands of cells. Microcolonies are often the functional units of the bacterial existence in natural settings and in the context of disease. We will provide theoretical framework describing the bacterial microcolonies as active viscoelastic materials and discuss how this theory might be useful in eukaryotic systems such as organoids, tumour spheroids or clustering immune cells. Microcolonies may further develop into even more complex bacterial communities known as biofilms - there, bacteria embed themselves in the self-secreted extracellular matrix creating an analogue of multicellular tissues. We will outline some future research avenues deepening this analogy and illustrate it with an intriguing example of wound healing in bacterial biofilms.

BP 6.6 Mon 16:45 H16

Playing it safe: information constrains collective betting strategies — •Philipp Fleig[1,2] and Vijay Balasubramanian[2] — [1]Max Planck Institute for Medical Research, 69120 Heidelberg, Germany — [2]Department of Physics & Astronomy, University of Pennsylvania, Philadelphia, PA 19104, USA

Risk is an inherent part of life and biological functions are partly shaped by the need to reduce risk. Broadly, risk arises from stochastic interactions of an organism with its environment. Every time an organism displays a particular response or behaviour (e.g. expresses a phenotype or exhibits a certain immune response), it is placing a bet with potential impact on its biological fitness. The more precisely the statistics of the environment are known to the organism, the more successfully bets can be placed. However, an organism typically has limited information about the statistics of the environment. This limitation should be accounted for in the adaptation of biological functions to the environment. We develop a theoretical principle where information geometric model complexity guides stochastic biological functions towards less risky betting strategies. In the framework of Bayesian inference, we show that given finite information about the environment, there is an optimally safe adaptation strategy set by the Bayesian prior. Furthermore, in a toy model of stochastic phenotypic switching by bacteria, we demonstrate how the implementation of our principle of "playing it safe" increases the fitness (population growth

rate) of the bacterial collective. We suggest that the principle applies broadly to problems of adaptation, learning and evolution.

BP 6.7 Mon 17:00 H16

Quantification of intracellular information flow — •Mirna Kramar[1], Mathieu Coppey[1], Thierry Mora[2], and Aleksandra Walczak[2] — [1]UMR 168, Institut Curie, Paris — [2]Laboratoire de Physique, Ecole Normale Supérieure, Paris

Signalling pathways are cascades of biochemical reactions which transduce signals from the exterior to the interior of the cell. By essence, these pathways convey information about the outside world which cells collect and process to adapt and guide decisions. The cell's ability to govern its functions correctly and precisely while relying on these intricate biochemical networks is surprising given the crowded and noisy cell interior, which indicates that the mechanisms cells use to process information are highly sophisticated. While our understanding of the constituents of the cellular machinery and the processes taking place in the cell is steadily increasing, little is known about the information flow within the cell. Are pathways conveying only on/off signals, or is there more graded information being transduced?

Here, we measure and quantify the information relayed through the MAPK signalling pathway, one of the key signalling pathways in eukaryotic systems. Using a synergy of an optogenetic experimental setup and a data analysis pipeline based on information theory, we quantify the input-output relationships within the MAPK signalling pathway. We show that the capacity of the pathway far exceeds the 1-bit value (on/off), and that collective systems of cell seem to exploit this capacity.