DY 22: Posters - Statistical Physics of Biological Systems

Time: Tuesday 14:00–16:00 Location: P1A

DY 22.1 Tue 14:00 P1A

Generic transport mechanisms for molecular traffic in cellular protrusions — •Isabella Krämer and Erwin Frey — Arnold Sommerfeld Center for Theoretical Physics, Ludwig-Maximilians-Universität, München, Deutschland

Transport of molecular motors along protein filaments in a half-closed geometry is a common feature of biologically relevant processes in cellular protrusions. Using a lattice-gas model we study how the interplay between active and diffusive transport and mass conservation leads to localised domain walls and tip-localisation of the motors. We identify a mechanism for task sharing between the active motors (maintaining a gradient) and the diffusive motion (transport to the tip), which ensures that energy consumption is low and motor exchange mostly happens at the tip. These features are attributed to strong nearest-neighbour correlations that lead to a substantial reduction of active currents, which we calculate analytically using an exact moment-identity.

DY 22.2 Tue 14:00 P1A

The architecture of bacterial biofilms depends on biofilm age and active force generation — •Anton Welker, Nadzeya Kouzel, and Berenike Maier — Biophysik, Köln, Deutschland

Many bacterial species aggregate in communities, called biofilms. In contrast to individual bacteria, bacterial biofilms show a complex structure, which makes them resistant against a variety of environmental factors and causes environmental, industrial and medical problems. Bacteria can adjust the three dimensional structure of their biofilms to varying environmental conditions. However, the molecular mechanisms governing biofilm structure are unclear. Here, we characterized the three-dimensional structure of biofilms formed by the human pathogen *Neisseria gonorrhoeae* at cellular resolution. In particular, the local density distribution, radial distribution function and parameters describing ordering and defects were implemented. We found that force-generating and force-defective bacterial microcolonies show significant differences in microcolony shape and density. The radial distribution function showed a significant difference in local ordering dependent on biofilm age, indicating that the density and the fraction of diplococci increased with biofilm age.

DY 22.3 Tue 14:00 P1A

Interference of deleterious and beneficial mutations in spatial habitats — •Philipp Klatt and Joachim Krug — Institute for Theoretical Physics, Cologne, Germany

One of the fundamental questions of population genetics is that of the rate at which beneficial or deleterious mutations are generated and incorporated into asexual populations. The quantity which describes this process is the speed of evolution. We here study a spatially structured model in which individuals of a population only compete locally on the time scale of a generation. In contrast to well-mixed models, where individuals compete with the whole population, the speed of evolution tends to a finite value in the limit of infinite habitat size when all mutations are either beneficial or deleterious [1,2]. We consider the general case where both types of mutations are present and map out the dependence of the speed of evolution on several parameters by interpreting analytical and numerical results. In contrast to the well-mixed case, we find that large populations undergoing a fitness decline caused by the accumulation of deleterious mutations (Muller's ratchet) cannot be rescued by a small rate of beneficial mutations. Moreover, the effects of deleterious and beneficial mutations on the speed of evolution are generally not additive, suggesting a nontrivial interference between the two types of mutational effects.

[1] Martens, E.A. and Hallatschek, O. (2011). Interfering waves of adaptation promote spatial mixing. Genetics 189:1045-60.
[2] Otwinowski, J. and Krug, J. (2014). Clonal interference and Muller's ratchet in spatial habitats. Physical Biology 11:056003.

DY 22.4 Tue 14:00 P1A

Recombination and Speciation on Fitness Landscapes — •Alexander Klug and Joachim Krug — Institut für Theoretische Physik, Universität zu Köln, Germany

Deterministic evolution models with selection, mutation and recombination display multiple stable stationary states in one realisation of a fitness landscape [1]. These distinct stationary states are mostly sharply peaked, which implies that the major part of the population of one stationary state has the same genotype, even in the presence of mutation. The populated genotypes of different stationary states can then be thought of as different species, because they differ in a number of loci and are stable. We are investigating various aspects of these stationary states, such as the number of distinct stationary states that exist in different landscapes models (House-of-Cards model, percolation model [2]) and the properties of their basins of attraction under the deterministic dynamics. The results are interpreted in the context of the concepts of Dobzhansky-Muller incompatibility [1] and mutational robustness [3].

[1] T. Paixao, K. E. Bassler, R. B. R. Azevedo, bioRxiv 008268
[2] S. Gavrilets, Trends Ecol. Evol. 12:307-12 (1997)
[3] E. van Nimwegen, J. P. Crutchfield, M. Huynen, PNAS 96:9716-9720 (1999)

DY 22.5 Tue 14:00 P1A

Non-equilibrium dynamics of heterogeneous biological systems — •Federica Mura and Chase Broedersz — Department of Physics, Ludwig-Maximiliams-University Munich, Theresienstrasse 37, 80333 Munich, Germany

Recent experiments indicate that many biological systems, including the cytoplasm, actin-myosin networks, and chromosomal loci can be driven out of thermodynamic equilibrium. The active dynamics of these systems is governed by local stochastic forces resulting from enzymatic processes. To provide insight into the non-equilibrium dynamics of such systems, we propose a simple stochastic model of a d-dimensional bead-spring network subject to a heterogeneous distribution of random driving forces. By using a combination of numerical simulations and analytical results we investigate how this non-equilibrium setting affects the system's steady state dynamics. We discuss how detailed balance is broken in the stochastic dynamics of different degrees of freedom and use this to quantify the rate of entropy production. Ultimately, this model could help to establish a systematic and more general method to extract information from a trajectory analysis of the stochastic dynamics of biological systems.

DY 22.6 Tue 14:00 P1A

Quantification of polymer loop shapes in application to chromosome oscillations — •Wenwen Huang and Vasily Zaburdaev — Max Planck Institute for the Physics of Complex Systems, Dresden, Germany

In this contribution, we model the chromosomes in meiotic fission yeast by pinned bead-rod loops in external force field. The 3D gyration tensor containing information of all beads positions is calculated. Based on the gyration tensor, the shape of polymer loops is quantified under different strength of the external force field. We show that the resulting shape is more rod-like and prolate under strong force and more sphere-like and oblate under weak force. Our study provides a quantitative description of the shape of pinned polymer loops under external field and may help us to describe the relevant biological processes in fission yeast such as chromosome oscillations and their alignment during meiosis.

DY 22.7 Tue 14:00 P1A

From flexible to stiff: a homopolymer model state diagram and its morphologies — •Benno Werlich and Wolfgang Paul — Institut für Physik, MLU Halle-Wittenberg, Germany

A hard-sphere homopolymer model with attractive interaction potential can be varied in its stiffness by modification the bondlength. According to this variation, thermodynamic functions and geometric observables undergo changes which are presented in a state-diagram for a chain length of N=40. The morphologies, e.g. in the low temperature range, show peculiar changes in monomer ordering towards stiffer chains. We use a Stochastic-Approximation Monte-Carlo (SAMC) method for our off-lattice model simulations and generate an estimation to the microcanonical entropy S(E). This entropy is our statistical weight for canonical production runs.

DY 22.8 Tue 14:00 P1A

Predictability of mutational trajectories in evolutionary rescue — •Jan Schmidt and Joachim Krug — THP, Cologne

Predictability of evolutionary pathways has been considered in terms of the strong selection weak mutation model (SSWM), where the whole population moves monomorphically along pathways with increasing fitness while maintaining a constant population size [1,2]. We compare these trajectories to pathways that are taken by a population which is on the verge of extinction. Here, conceptually, the assumption of SSWM is no longer valid. Dropping the constraint of fixed population size, where the population does not fixate the intermediate types before the rescuing type is reached, this process is described in terms of a branching process [3].

[1] D.M. Weinreich et. al., Science 312, 111 (2006)
[2] J. Franke et. al., PLoS Comput Biol 7, e1002134 (2011)
[3] B. Bauer and C. Gokhale, Scientific Reports 5, 9607 (2015)

DY 22.9 Tue 14:00 P1A

Local optima in NK fitness landscapes — •Benjamin Schmiegelt, Sungmin Hwang, and Joachim Krug — Institute for Theoretical Physics, University of Cologne

Fitness landscapes, the assigment of fitness values to genotypes, determine the structural impact of selection on population dynamics. Populations will, especially under strong selection pressure, cluster around local optima. The number of local optima is also considered a measure of ruggedness, complexity and difficulty for a population to move on the landscape. The NK model models landscapes with parameter-controlled ruggedness and many possible interaction schemes between loci, sharinf similarities with p-spin glass models. For a quasi-one dimensional circular interaction layout the expected number of local optima is well established. We consider instead the case of random interaction networks, mean field and other variations and find, contrary to traditional assumptions, quantitatively different asymptotics for the expected numbers of local optima from the circular case.

[1] Weinberger, E. D. (1991). Local properties of Kauffman's N-k model: A tunably rugged energy landscape. Physical Review A, 44(10), 6399.

[2] Limic, V., & Pemantle, R. (2004). More rigorous results on the Kauffman-Levin model of evolution. The Annals of Probability, 32(3), 2149-2178.

[3] Schmiegelt, B., & Krug, J. (2014). Evolutionary accessibility of modular fitness landscapes. Journal of Statistical Physics, 154(1-2), 334-355.

DY 22.10 Tue 14:00 P1A

Stochastic switching of of Min proteins in short Escherichia coli cells — •Lukas Wettmann[1] and Karsten Kruse[2] — [1]Theoretische Physik, Saarland University, Saarbrücken, Germany — [2]NCCR Chemical Biology, Department of Biochemistry, Department of Theoretical Physics, University of Geneva, Geneva, Switzerland

Intracellular processes are subjected to noise, be it through thermal fluctuations or, for example, molecular noise. The latter case is especially true when the total number of involved molecules inside the cell is low. More interesting than the, for the case of gene expression, local changes in protein number is the stochastic behavior of spatially inhomogeneous protein distributions inside the cell.

To this end, we study the influence of noise on the dynamics of the Min system. The Min proteins are a family of proteins which through self-organization are able to exert spatial pole-to-pole oscillations in rod-shaped E. coli cells. These oscillations cause the division site to be located along the symmetry axis of the cell, ensuring equal-sized daughter cells. In contrast, for short cells, the oscillations are replaced by stochastic switching of the proteins between two stable polar configurations. This behaviour can cause the emergence of mini cells if the residence times are sufficiently long.

We developed a mechanism based on the underlying molecular processes to study the dynamics of the Min proteins. With this mechanism, we are able to use a framework developed in earlier work to analyze the behaviour of the Min proteins in the limit of weak noise and calculate the residence times as a function of the cell length.