DY 41 Dynamical Physics in Biological Systems

Time: Thursday 10:00-11:30

DY 41.1 Thu 10:00 SCH 251 DNA as a rigid-body chain — •NILS BECKER and RALF EVERAERS

— MPI Physik Komplexer Systeme, Dresden The functioning of the DNA molecule in many biological situations depends on its sequence-dependent elastic behavior on a scale of a few base pairs to a few helical turns. Examples include winding onto nucleosomes and transcription regulation.

We investigate the elastic properties of DNA at this scale. We study a chain model in which the monomers are the base pairs, considered as rigid bodies. We take into account the full detail of their sequence dependent nearest neighbor interaction, including coupling of rotational and translational degrees of freedom, within a harmonic approximation.

This model fits in as a natural step in a coarse-graining hierarchy of DNA models. On the microscopic side, it can be parametrized by existing inter-base pair harmonic potentials. This allows a calculation of the sequence-dependent elastic response to forces and moments that are applied to short stretches of DNA. Towards larger scales, by taking an appropriate continuum limit, we can relate it to classic chain models for semiflexible polymers.

DY 41.2 Thu 10:15 SCH 251

A Bayesian Approach to the Evaluation of Dynamic Force Spectroscopy Experiments — •SEBASTIAN GETFERT, PETER REIMANN, and MARTIN RAIBLE — Condensed Matter Theory, Universität Bielefeld, Universitätsstraße 25, 33615 Bielefeld

The components of a biomolecular complex can be connected via suitable linkers to the tip of an atomic force microscope and a moving surface. When this surface is pulled apart at constant velocity the force f that acts on the bond increases (approximately) linearly in time until the chemical bond ruptures. This process is of stochastic nature. The distribution of the rupture forces and in particular the maximum depends in a characteristic way on the loading rate \dot{f} and on binding parameters like dissociation length and (force-free) dissociation rate [1]. Thus the evaluation of rupture force data from dynamic force spectroscopy experiments allows to draw conclusions about the energy landscape of the bond. We present a Bayesian approach to this evaluation in which experimental uncertainties can be included in a natural way. Further we discuss from a statistical point of view to which degree of accuracy the parameters of the distribution can be determined.

[1] E. Evans and K. Ritchie, Biophys. J. 72, 1541 (1997)

DY 41.3 Thu 10:30 SCH 251

Neuronal Growth: A Bistable Stochastic Process — •TIMO BETZ, DARYL LIM, and JOSEF KÄS — Institut for Soft Matter Physics, University of Leipzig, Linnestr. 5, 04103 Leipzig, Germany

During the past decade, modeling biological systems as stochastic processes has given tremendous insight into nature's working principles at the level of networks, single cells, and molecules. However, the stochastic nature of neuronal growth has hardly been investigated. The basic step in the correct neuronal wiring of a developing organism is the controlled advancement of a highly motile structure, called the growth cone, which is directed by gradients of chemical guidance cues. We report on the first statistical analysis of the stochastic fluctuation of a neuronal growth cone's leading edge movement. Describing the edge movement with a stochastic process allows inferring a bistable potential from the edge velocity distribution. Using Kramers' approach to calculate decay rates, an isotropic noise parameter can be determined, which we used to consistently connect the measured edge velocity distribution and the residence time distribution. An according analysis of the growth cone's motility confirms the model, and predicts that linear changes of the bistable potential might result in the directed growth cone translocation. These results help to understand how the growth cone can detect chemical gradients that are on the order of one molecule across its diameter, even in the highly noisy environment of a developing organism.

DY 41.4 Thu 10:45 SCH 251

The emergence of Ca^{2+} puffs as intracellular escape process — •RÜDIGER THUL and MARTIN FALCKE — Hahn-Meitner Institut, Abteilung Theorie, Glienickerstrasse 100, 14109 Berlin

Recent experiments and theoretical investigations have demonstrated that intracellular Ca^{2+} is a stochastic nonlinear medium. Local and

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global patterns are exclusively driven by fluctuations. These patterns form a hierarchy. Global events are built from a series of local incidents, which are termed Ca^{2+} puffs. A detailed study of Ca^{2+} puffs reveals that their emergence can be mapped to an escape process. That permits an analytic calculation of the mean stochastic fraction of the puff period. A quantification of the leading time scales provides further insights into this escape process. Moreover, we show that the spatial restriction of Ca^{2+} puffs enforces a discrete modeling of the Ca^{2+} dynamics.

DY 41.5 Thu 11:00 SCH 251

Discrete Model for Pattern Formation in Bacterial Colonies — ●PAWEL ROMANCZUK¹, UDO ERDMANN², HARALD ENGEL¹, and LUTZ SCHIMANSKY-GEIER² — ¹Institut für Theoretische Physik, Technische Universität Berlin, Hardenbergstr. 36, 10623 Berlin, Germany — ²Institute für Physik, Humboldt Universität zu Berlin, Newtonstr. 15, 12489 Berlin, Germany

Bacterial colonies of Escherichia coli and Salmonella typhimurium show complex patterns of high density cell aggregates when exposed to certain nutrients. Decisive for this pattern formation is the production of a potent chemoattractor by the bacteria as a reaction to the nutrient [1]. The observed bacterial patterns range from temporary spots formed in liquid medium to "sunflower like" spot arrangements of striking complexity in a semi-solid medium. Motivated by this observations we suggest a simple model for the description of bacterial colonies based on the concept of Active Brownian motion [2]. Our model represents an interesting alternative to the usually employed "pure" reaction-diffusion equations as it allows us to study the macroscopic pattern formation of the colony, the mesoscopic dynamics of bacterial ensembles (swarming), as well as the microscopic dynamics of single cells. Here we will present the obtained qualitative and quantitative numerical results of our model and compare them with the experimentally observed bacterial dynamics. [1] Budrene, E. O. und H. C. Berg: Dynamics of formation of symmetrical patterns by chemotactic bacteria. Nature, 376:49-53, 1995.

[2] Erdmann, U., Kollektive Bewegung, Logos Verlag, Berlin, 2004.

DY 41.6 Thu 11:15 SCH 251

Dynamics of epidemic outbreaks in heterogeneous populations — •ALEJANDRO MORALES GALLARDO, DIRK BROCKMANN, LARS HUFNAGEL, and THEO GEISEL — MPI for Dynamics and Self-Organization, Göttingen, Germany

The dynamics of epidemic outbreaks have been investigated in recent years within two alternative theoretical paradigms. Among the most successful models is the deterministic susceptible-infected-recovered (SIR) model which approximately describes the dynamics for a large number of individuals and in which homogeneous contact rates are assumed. The central parameter of the SIR model is the basic reproduction number. the average number of secondary infections caused by one infected individal. Recently, scale free network models have received much attention as they account for the high variability in the number of social contacts involved. These models predict an infinite basic reproduction number in some cases. We investigate the impact of heterogeneities of contact rates in a generic model for epidemic outbreaks. In constrast to common static network models we investigate a system in which both the time periods of being infectious and the time periods between transmissions are Poissonian processes. The heterogeneities are introduced by means of strongly variable contact rates which yield power laws in the number of overall contacts. In contrast to scale free network models we observe a finite basic reproduction number and, counterintuitively a smaller overall epidemic outbreak as compared to the homogeneous system. Our study thus reveals that heterogeneities in contact rates does not facilitate the spread to infectious disease but rather attenuates it.