

## BP 18: Poster VI

Cell Adhesion and Migration &amp; Multicellular Systems (BP 16.1 – BP 16.27)

Time: Tuesday 14:00–16:00

Location: P2/2OG

BP 18.1 Tue 14:00 P2/2OG

**Altering the early development of *Caenorhabditis Elegans* via laser ablation** — •VINCENT BORNE, PHILIPP STRUNZ, and MATTHIAS WEISS — Experimental Physics I, University of Bayreuth, Germany

While the role of biochemical signaling during embryonic development has been extensively studied, the role of mechanical cues in this process are still only partially understood. For the early development of the model organism *Caenorhabditis elegans*, mechanical forces have been shown to be key for a proper cell arrangement in the embryo until gastrulation: Based on mutual pushing forces of cells within the confining egg shell, a simple relaxation model was shown to be capable of predicting cell trajectories and final positions. Here we have probed the validity of this model when perturbing the nematode's embryogenesis at very early stages via laser microsurgery. Experimental results observed after ablating early precursor cells suggest that an extension of the model, e.g. updating the cortical stiffness of cells for reproducing correct wetting angles between neighboring cells, is required. Preliminary results on developing such an extended model, based on dissipative particle dynamics simulations, are discussed.

BP 18.2 Tue 14:00 P2/2OG

**Morphodynamics in the Foraging of *Physarum polycephalum*** — •LISA SCHICK, MIRNA KRAMAR, and KAREN ALIM — Max Planck Institute for Dynamics and Self-Organization, Göttingen, Germany

Foraging behaviour of animals is generally described as optimized for maximal energy uptake per time spend foraging within optimal foraging theory. Food sources often occur as food patches, so that foraging becomes a balance between time spent for exploration and time spent for patch exploitation leading to the question at which point a patch should be abandoned. Foraging behaviour in a patchy habitat can also be observed in unicellular but spatially extended organisms like *Physarum polycephalum*. However, it is unclear which foraging strategy the large and adaptive network-like morphology allows for. The plasmodial network of *P. polycephalum* adapts its morphology in the process of foraging by mass transport. Recent observations show that on encounter of a food patch, depending on body size, the whole body is relocated for exploitation. We here study the morphological changes as a function of network size and nutritional state by introducing a model for the exploration and exploitation phases in *P. polycephalum*. We estimate the energy uptake from our foraging observations in order to obtain rules for the foraging behaviour.

BP 18.3 Tue 14:00 P2/2OG

**Network adaptation to transient stroke** — •LEONIE BASTIN<sup>1</sup>, KOMAL BHATTACHARYYA<sup>1</sup>, and KAREN ALIM<sup>1,2</sup> — <sup>1</sup>Max Planck Institute for Dynamics and Self-Organization, Göttingen, Germany — <sup>2</sup>Technical University of Munich, Germany

Damage is a risk that all kinds of networks are exposed to. This includes overload in power grids as well as the formation of blood clots in brain microvasculature. The occlusion of a single blood vessel in the brain can cause severe tissue damage and cognitive dysfunction. Experiments and simulations have been done in the past to understand the effect of vessel occlusions on brain blood flow and network architecture. Here, we are interested in the morphological adaptation of a network to occlusion induced flow changes. Is it possible that networks reorganise to minimise the damage caused by lack of flow? For this aim, we use the model organism *Physarum polycephalum*. In the plasmodial stage, the slime mould forms a network of interconnected tubes through which its cytoplasm streams back and fro, driven by peristaltic tube contractions. Previously, morphological changes due to changes in the contraction pattern have been observed. Here, we locally induce stalling of the flow in a tube of the network and analyse the effect of flow changes on network morphology.

BP 18.4 Tue 14:00 P2/2OG

**Modelling cell monolayers with an elastic phase field approach** — •ROBERT CHOJOWSKI, ULRICH S. SCHWARZ, and FALKO ZIEBERT — Institute for Theoretical Physics and BioQuant, Heidelberg University, Germany

Motion and force generation of cell collectives are crucial in many biological processes, including wound healing and cancer metastasis. The first case can be quantitatively investigated in the so-called wound healing assay, when 2D cell monolayers start to move into empty space after removal of a barrier, often by forming protrusions led by so-called leader cells. During recent years it has become clear that these cell monolayers are both dynamic and elastic at the same time, a combination which is hard to model with conventional approaches. Here we introduce a new model that uses phase fields for the dynamical aspects and the theory of thin elastic sheets for the mechanical aspects. Our continuum equations can be solved numerically by a combination of spectral and matrix methods, and they can be compared to analytical solutions. We demonstrate how our approach works for several paradigmatic situations, namely contraction of a 1D bar and a 2D disc, and formation of elastic protrusions for a 2D wound healing situation due to localized forces at the interface.

BP 18.5 Tue 14:00 P2/2OG

**Stochastic Cell-Cell Interactions in Confined Systems** — •NICOLAS ARLT<sup>1</sup>, DAVID BRÜCKNER<sup>1</sup>, ALEXANDRA FINK<sup>2</sup>, JOACHIM RÄDLER<sup>2</sup>, and CHASE BROEDERSZ<sup>1</sup> — <sup>1</sup>Arnold-Sommerfeld-Center for Theoretical Physics and Center for NanoScience, LMU Munich — <sup>2</sup>Faculty of Physics and Center for NanoScience, LMU Munich

Assemblies of motile cells can exhibit distinct behaviors, such as clustering to heal a wound or dispersal in cancer cell invasion. Such collective behaviors have been shown to be intimately related to the local contact interactions between individual cells. Here, we investigate how cell-cell interactions affect the dynamics of confined migrating cells. To this end, we place pairs of highly invasive MDA-MB-231 cancer cells on adhesive micropatterns, consisting of two square patches connected by a thin constriction. In this setup, we observe that the cells repeatedly transition across the constriction, leading to repeated 'cellular collisions' in a standardized microenvironment. By obtaining a large dataset of such collision trajectories, we infer an equation of motion for these interacting cell pairs. This model reproduces the key statistics of the interaction dynamics, such as position- and velocity correlations. Our approach allows us to characterize the cell-cell interactions, which we compare for different celltypes and different types of confinement.

BP 18.6 Tue 14:00 P2/2OG

**Collective cell migration in 3D micro-tumours** — •TOM BRANDSTÄTTER<sup>1</sup>, DAVID BRÜCKNER<sup>1</sup>, YU LONG HAN<sup>2</sup>, MING GUO<sup>2</sup>, and CHASE BROEDERSZ<sup>1</sup> — <sup>1</sup>Arnold-Sommerfeld-Center For Theoretical Physics and Center of NanoScience, LMU Munich, Germany — <sup>2</sup>Department of Mechanical Engineering, Massachusetts Institute of Technology, Cambridge, MA, USA

The coordinated migration of cell collectives is key to many biological processes, including cancer progression. Here, we study the dynamical behaviour of in vitro micro-tumours. Initially, these micro-tumours are in a benign phase, exhibiting strongly correlated and highly persistent collective rotation of nearly all cells inside the spheroid. We investigate local interactions that can induce this collective mode of cell migration, using a physical minimal model for cell migration.

To constrain our models of interacting active particles, we analyse experimental data obtained from in vitro micro-tumours of various sizes. These experimental observations, including correlation functions and spatio-temporally resolved measures of the local persistence, place strong constraints on possible candidate models.

Furthermore, we find that these benign tumours eventually transition to a malignant state, which is characterized by an unconfined, non-definite shape with branches invading the surrounding tissue. Collective rotation is completely lost and the cancer cells show correlated but disordered motion. We aim to understand this drastic change in the collective behaviour in terms of the changes in single-cell behaviour and in cell-cell interactions.

BP 18.7 Tue 14:00 P2/2OG

**Unravelling the biomolecular mechanism of light-switchable adhesion of *Chlamydomonas* to surfaces** — •ANTOINE GIROT, RODRIGO CATALÁN, ALEXANDROS FRAGKOPOULOS, ANAËLLE CHRÉTIEN, LINE HOLTZER, and OLIVER BÄUMCHEN — Max Planck Institute

for Dynamics and Self-Organization (MPIDS), Am Fassberg 17, 37077 Göttingen, Germany

Bioadhesion is a ubiquitous phenomenon for many living systems such as mussels, bacteria or microalgae. In this work, we focus on the adhesion of the flagellated microalga *Chlamydomonas reinhardtii*. We discovered that *Chlamydomonas* exhibits light-switchable adhesion, in which the flagella of the cells stick to surfaces under blue but not under red light. Our goal is to unravel the biomolecular mechanism of this specific light-regulated behaviour. In order to characterise the adhesiveness of *Chlamydomonas* to surfaces, two different experimental approaches are carried out. First, the kinetics of the adsorption and desorption of a cell suspension to a surface in response to a light switch is recorded. Second, *in vivo* micropipette force spectroscopy experiments are performed to precisely measure the adhesion force of single cells. By applying these methods for different wild-type as well as genetically modified strains, we aim at identifying characteristic gene sequences associated to the cells adhesiveness. To unravel the blue-light photoreceptor that triggers the light-switchable adhesion, experiments with specific photoreceptor-deleted mutants are performed. Finally, we also investigate how the glycosylation of the flagella membrane proteins affects the adhesiveness of *Chlamydomonas*.

BP 18.8 Tue 14:00 P2/2OG

**Mechanisms behind growth and locomotion of *Physarum polycephalum*** — ●NICO SCHRAMMA<sup>1</sup> and KAREN ALIM<sup>1,2</sup> — <sup>1</sup>Max Planck Institute for Dynamics and Self-Organization — <sup>2</sup>Technical University of Munich

The unicellular slime mould *Physarum polycephalum* is able to find and efficiently connect multiple food sources by reorganising its network-like morphology. Acto-myosin driven peristaltic contractions of the networks' tubes enable this efficient mass transport. Moreover, this organism is known to respond to a huge variety of stimuli by altering its body plan and the contraction patterns.

However, it is not understood how this slime mould moves and which mechano-chemical parameters are varied in order to modulate the contractions driving the migration. Here, we present a multi-timescale approach including brightfield-, birefringence- and fluorescence microscopy, particle image velocimetry, and a non-invasive air-jet indentation method to observe the contraction patterns, acto-myosin organisation, local flow-profile and spatial variations in cortex elasticity in small *Physarum* during migration.

BP 18.9 Tue 14:00 P2/2OG

**Energetic cost of morphological states of *Physarum polycephalum*** — ●LEONIE KEMETER<sup>1</sup>, MIRNA KRAMAR<sup>1</sup>, and KAREN ALIM<sup>1,2</sup> — <sup>1</sup>Max Planck Institute for Dynamics and Self-Organization — <sup>2</sup>Technical University of Munich

An interconnected network of tubes forms the flow-driven plasmodial networks of the unicellular slime mould *Physarum polycephalum* and provides an astonishing example of self-organization in biological systems: the tubes' oscillatory activity is responsible for driving the flow of endoplasm through the network, thus forming the morphology via the contractions. Changes in oscillations and the resulting morphological changes help *Physarum* react to its environment. For example, it increases effective Taylor dispersion by pruning of small tubes during foraging. Recent experiments show two distinct morphological states - one fan-like slower state and one lightning-like faster state. Interestingly, the slime mould seems to switch randomly between the two states. Knowing the energetic cost of those morphological states would allow for possible explanations for this behaviour, e.g. a high energetic cost of the lightning strike suggests that it is used only when advantages of this state such as the higher speed are necessary. We model the elastic and dispersive energy of a network showing both morphological states. The model uses the tried and tested approach that the tube's contractions result in a peristaltic wave across the network and needs only the time-averaged radii of the tubes as input from the experiments. The modelled contractions can later be compared to the measured contractions to check the validity of the model.

BP 18.10 Tue 14:00 P2/2OG

**The effect of blue light on *Physarum polycephalum* cytoplasm flow for different topologies** — ●SIYU CHEN<sup>1</sup>, FELIX BÄUERLE<sup>1</sup>, JEAN-DANIEL JULIEN<sup>1</sup>, and KAREN ALIM<sup>1,2</sup> — <sup>1</sup>Max Planck Institute for Dynamics and Self-Organization, Göttingen, Germany — <sup>2</sup>Technische Universität München, München, Germany

The network-forming unicellular slime mould *Physarum polycephalum*

adapts to external stimuli in a coordinated manner over an extended body plan in space and time. This is achieved by sharing information and resources across the body plane in a peristaltic wave that adapts to body size.

Among the common external stimuli, one of the most studied stimuli is blue light, which triggers a retreat response in *P. polycephalum*. This change of the slime mould's environment are met with an adaptation of its tubes' oscillatory activity.

In this project we focus on the relationship between the topology of the network and their responses towards the external stimuli. We suspect that, when *P. polycephalum* was exposed to the blue light, it regulates the fluid properties of the endoplasm, and this process diversifies with different topology. Here we experimentally observe how *P. polycephalum* in different topologies responses to blue light stimuli. We quantify its contraction pattern and flow information such as velocity and viscosity to compare with our theoretical expectations.

BP 18.11 Tue 14:00 P2/2OG

**Flow reorganisation in the brain microvasculature during a stroke** — ●AGNESE CODUTTI<sup>1</sup> and KAREN ALIM<sup>1,2</sup> — <sup>1</sup>Max Planck Institute for Dynamics and Self-Organization, 37077 Göttingen, Germany — <sup>2</sup>Physik-Department, Technische Universität München, Garching, Germany

Ischemic brain strokes are a major concern for public health. Major strokes obstructing main arteries cause reorganisation of flows throughout the microvasculature, likely with a long lasting influence on brain microvasculature behaviour and topology. Strikingly flow reorganisation is different in different parts of the brain vasculature. While the loopy surface arteriole network undergoes stops and reversals during a stroke, the penetrating arterioles exhibit steady flow direction. One hypothesis is that the flow reorganisation at the surface prevents changes in the penetrating arterioles. Here, we investigate if network topology and hierarchy of the system drives penetrating arterioles to be robust asking: Is the network designed to be resilient to such changes? To test this hypothesis, we analytically solved the flows in a toy model of an H-shaped network module showing that flow reversal in the penetrating arterioles can happen in principle for great pressure instabilities, due to the hierarchy and topology of the network. In two dimensional irregular networks optimised for transport, the pressure difference needed for the reversal yet is even higher, supporting the hypothesis of the in-built resilience of the network. Currently, we are testing the dependence on different network properties to identify the main factor inducing the resilient behaviour.

BP 18.12 Tue 14:00 P2/2OG

**Parameter Optimization of a Cellular Potts Model using Multiple Micro-Pattern Cell Migration Experiments** — ●SOPHIA ANNA SCHAFFER<sup>1</sup>, ANDRIY GOYCHUK<sup>2</sup>, CHRISTOPH SCHREIBER<sup>1</sup>, ERWIN FREY<sup>2</sup>, and JOACHIM RÄDLER<sup>1</sup> — <sup>1</sup>Fakultät für Physik, Ludwig-Maximilians-Universität, Geschwister-Scholl-Platz 1, 80539 München — <sup>2</sup>Arnold Sommerfeld Center, Ludwig-Maximilians-Universität, Theresienstraße 37, 80333 München

Modelling cell migration is important to simplify and understand the highly complex machinery of cells. Cellular Potts Models (CPMs) have been successfully used to reproduce cell migration patterns in two dimensions. They are capable to reproduce cell behavior with increasing levels of complexity and biological accuracy from single cells to collective migration. A particular challenge for computational simulations is to optimize model assumptions and parameters so that many experimental data sets are fitted simultaneously. Cells in confining geometries show restricted motion and various properties of cell behavior such as deformation, persistence, adhesion and polarization can be separately probed. Here, we still present a rational approach to use a set of geometries with complementary properties to determine parameter values successively in a systematic manner using one specific cell line. The general concept is to determine model parameters successively by using the emergent properties of the different geometry designs.

BP 18.13 Tue 14:00 P2/2OG

**Evaluation of cancer spheroid growth in synthetic hydrogels with light sheet microscopy** — ●VIKTORIA ZIEGER, TIMO BETZ, and BART E. VOS — Institute of Cell Biology, ZMBE, Münster, Germany

Tumours of cancer cells are in constant interaction with their surroundings, where physical forces are applied to the extracellular matrix (ECM) to eventually allow the cancer cell to migrate and to form

metastasis. We evaluate cancer spheroid growth and cell invasion over time as a function of the rigidity of a non-linear ECM. To achieve this, the extracellular matrix is mimicked by a synthetic hydrogel, which is biocompatible, strain-stiffening and can be finely mechanically tuned without affecting the structural properties. This allows control of the physical properties and the mechanoenvironment.

To gain 3D timeseries of spheroid evolution as well as hydrogel deformation, an in-house scanning light-sheet microscope is built, which allows rapid imaging while reducing photo-bleaching for long-time measurements. To avoid sample motion, the set-up combines axial light-sheet movement via a scanning galvanometer mirror system and simultaneous focus adjustment via an electrically tunable lens. This prevents the necessity to move the sample and enables precise time-resolved 3D images of spheroid growth and cell invasion.

BP 18.14 Tue 14:00 P2/2OG

**The effect of lidocaine and calcium signaling on the adhesion of *Chlamydomonas* to surfaces** — ●MARZIEH KARIMI, HENNING LÜHRS, ANTOINE GIROT, and OLIVER BÄUMCHEN — Max Planck Institute for Dynamics and Self-Organization (MPIDS), 37077 Göttingen, Germany

Photosynthetic microorganisms are ubiquitous in nature and applied in photobioreactor technologies for molecular farming and as a sustainable source of biofuels. However, the formation of microbial biofilms in such photobioreactors appears as a major technological issue, which could be solved by tuning the adhesion of the cells. In this context, we discovered that the adhesion of the microalga *Chlamydomonas reinhardtii* to surfaces is switchable by light (Kreis et al., Nature Physics, 2018). Under blue light, the cells stick to surfaces, while under red light they do not show any adhesion. Previous experiments suggest that this change in adhesion is based on the translocation of the adhesion-mediating protein FMG-1B within the flagella membrane. In this work, we study the influence of lidocaine as a signal inhibitor on the light-switchable adhesion of *Chlamydomonas*. Using in vivo micropipette force spectroscopy experiments, we find that the light-switchable adhesion of the flagella is completely inhibited in the presence of lidocaine in any light condition. Since lidocaine is known to inhibit calcium ion channels, we hypothesize that such a photocurrent is involved in the signal transduction from the blue-light photoreceptor to the flagella membrane.

BP 18.15 Tue 14:00 P2/2OG

**Fluid flow controls morphological changes** — ●NOAH ZIETHEN<sup>1</sup> and KAREN ALIM<sup>1,2</sup> — <sup>1</sup>Max Planck Institute for Dynamics and Self-Organization, Göttingen, Germany — <sup>2</sup>Technical University of Munich, Garching, Germany

The morphology of biological transport networks is often regarded as a result of optimization under a given demand. As demands may change rapidly in life, biological flow networks continuously adapt. The shear rate inside the flow network is largely assumed to be the control mechanism of this adaptation. However, direct experimental evidence for this hypothesis is still missing, and the theoretical implication of such local adaptation on the network dynamics is not fully understood.

Here, the model organism *Physarum polycephalum* allows to directly test causality between flow shear rate change and vessel pruning. *P. polycephalum* forms a network of connected tubes exhibiting a complex oscillatory shuttle streaming inside them. We image and quantify the time evolution of single vessels in *Physarum* by extracting the vessel diameter evolution and the corresponding flow field using particle image velocimetry. These measurements reveal a time-delayed response in the tube diameter trend on the average flow magnitude, which results in some data sets in a pruning behavior and for others in an oscillatory interplay between the two quantities. Motivated by the experimental result, we build a feedback model taking into account the local minimization of energy dissipation and the coupling to the network, which is able to reproduce the bistability found in the data.

BP 18.16 Tue 14:00 P2/2OG

**Quantification of the collective dynamics of endothelial cells in a two-dimensional confluent layer** — ●ANSELM HOHLSTAMM, MATS LEIF MOSKOPP, ANDREAS DEUSSEN, and PETER DIETERICH — Institut für Physiologie, Medizinische Fakultät, TU Dresden

Collective dynamics of confluent cells results from a complex interplay of single cell dynamics and cell-to-cell interactions. In order to better understand these processes, a quantitative analysis of cell movements is essential. Hence, we aim to quantify the migration activity of single cells influencing the whole cell collective. Human umbilical

vein endothelial cells (HUVECs) were seeded and stained with low concentrations of a fluorescent dye. The cells were observed for up to 48 hours via time-lapse microscopy (dt = 10 min) and trajectories were obtained with an in-house developed image processing software. Typically, several 10.000 cell trajectories per experiment were detected within an area of 6 x 7 mm. HUVECs showed lively proliferation generating a confluent two-dimensional monolayer in a non-equilibrium situation. This process can be characterized by an exponential velocity distribution of the cells where the mean squared velocity showed a slow decrease. The mean squared displacement indicated a sub-diffusive behaviour. This is consistent with an increasing cell density forcing cells to localize their positions. Besides, the spatial velocity correlation function showed an exponential-like decrease with correlation lengths of around 60  $\mu\text{m}$  ( $\sim 3$  cell diameters). In conclusion, we quantified the dynamics of confluent cells allowing us to evaluate the effects of humoral or pharmaceutical stimulations in future.

BP 18.17 Tue 14:00 P2/2OG

**The Physics of Carcinomas: A multi-scale analysis on primary tumor tissues** — ●FRANK SAUER<sup>1</sup>, STEFFEN GROSSER<sup>1</sup>, ERIK W. MORAWETZ<sup>1</sup>, THOMAS FUHS<sup>1</sup>, FREDERIC RENNER<sup>1</sup>, BENJAMIN WOLF<sup>3</sup>, SONJA KALLENDRUSCH<sup>4</sup>, HANNAH-MARIE SCHOLZ MARGRAF<sup>1</sup>, JÜRGEN LIPPOLDT<sup>1</sup>, HEIKO TZSCHÄTZSCH<sup>6</sup>, JÜRGEN BRAUN<sup>7</sup>, INGOLF SACK<sup>6</sup>, CLAUDIA T. MIERKE<sup>2</sup>, INGO BECHMANN<sup>4</sup>, SUSANNE BRIEST<sup>3</sup>, LARS-CHRISTIAN HORN<sup>5</sup>, MICHAEL HÖCKEL<sup>3</sup>, BAHRIYE AKTAS<sup>3</sup>, and JOSEF A. KÄS<sup>1</sup> — <sup>1</sup>Soft Matter Physics Division, Peter-Debye-Institute for Soft Matter Physics, Leipzig, Germany — <sup>2</sup>Biological Physics Division, Peter Debye Institute for Soft Matter Physics, Leipzig, Germany — <sup>3</sup>Department of Gynecology and Obstetrics, Universitätsklinikum Leipzig, Germany — <sup>4</sup>Institute of Anatomy, Universitätsklinikum Leipzig, Germany — <sup>5</sup>Institute of Pathology Ad Interim, Universitätsklinikum Leipzig, Germany — <sup>6</sup>Department of Radiology, Charité-Universitätsmedizin, Berlin, Germany — <sup>7</sup>Institute of Medical Informatics, Charité-Universitätsmedizin, Berlin, Germany

Cancer is a heterogeneous disease and most fatalities from solid tumors arise from their ability to systematically metastasize. This metastatic cascade is a multi-scale process. In cooperation with the University Hospital Leipzig, we are realizing a full-scale biophysical analysis on selected primary tumor tissue samples. Covering a range from macroscopic bulk properties down to single cell features, we are aiming at an interdisciplinary tumor characterization to contribute to a better understanding of the systemic nature of the disease cancer.

BP 18.18 Tue 14:00 P2/2OG

**Stick-slip motion for a cellular glider with translational and rotational friction** — ●PINTU PATRA<sup>1,2</sup>, ANNA BATTISTA<sup>1,2</sup>, and ULRICH S. SCHWARZ<sup>1,2</sup> — <sup>1</sup>BioQuant, Heidelberg University & University Medical School, Heidelberg, Germany — <sup>2</sup>Institute for Theoretical Physics, Heidelberg University, Heidelberg, Germany

Cellular movement over planar surfaces is often limited by sliding friction at the cell-substrate interface. A typical feature of sliding friction is stick-slip motion which consists of periods of slow (stick phase) and fast movement (slip phase). Stochastic multi-contact models explain the observed stick-slip motion at intermediate speeds of the translational motion. These models consider an ensemble of molecular bonds that form and rupture at the interface between two surfaces moving relative to each other. Here we extend this theoretical framework to include also the effect of rotational motion, a situation which arises e.g. for malaria parasites gliding on flat substrates. First, we derive analytical force-velocity and torque-angular velocity relations for pure translation and pure rotational driving, respectively. Then we show that a critical region of large bond fluctuations observed for the combined driving smoothly connects the corresponding regimes that exist for the purely translational and purely rotational cases. We also find an additional regime at small translational but high rotational velocities in which large deviations occur in the circularity of the cellular glider.

BP 18.19 Tue 14:00 P2/2OG

**One-dimensional active gel models for optogenetic control of cell locomotion** — ●OLIVER MAX DROZDOWSKI<sup>1,2</sup>, FALKO ZIEBERT<sup>1,2</sup>, and ULRICH SEBASTIAN SCHWARZ<sup>1,2</sup> — <sup>1</sup>Institute for Theoretical Physics, Heidelberg University, Philosophenweg 19, 69120 Heidelberg, Germany — <sup>2</sup>BioQuant, Heidelberg University, Im Neuenheimer Feld 267, 69120 Heidelberg, Germany

Cell motility is essential in all domains of life, including development,

wound healing and cancer. In order for a cell to start moving, a transition from a symmetric non-motile state to a polarized moving state has to occur. One-dimensional models of active gels based upon continuum mechanics have resulted in a quantitative understanding of how the interplay between contraction and flow in the actin cytoskeleton can lead to this transition. Recently, optogenetics has emerged as a promising experimental tool to control these processes. In this work we theoretically investigate if and how optogenetics could be used to control cell locomotion. We find that effects from external optogenetic signals can be incorporated in existing models as perturbations of solutions to the governing equations. We investigate an active Maxwell model with elastic boundary conditions, which cannot sustain the broken symmetry on its own, and show that asymmetric signals can lead to motility that then stops when the optogenetic activation ends. Additionally, we discuss models that also couple to a dynamic concentration field of motor proteins. Here optogenetic perturbations can both initiate and arrest steady states of motility. Together these results suggest that optogenetics could indeed be used to control cell movement.

BP 18.20 Tue 14:00 P2/2OG

**Spatio-temporal characteristics of eukaryotic single-cell motility on fibronectin coated micro-lanes** — ●JOHANNES HEYN, CHRISTOPH SCHREIBER, and JOACHIM RÄDLER — Faculty of Physics and Center for NanoScience, Ludwig-Maximilians-Universität München, Geschwister-Scholl-Platz 1, D-80539 Munich, Germany

Studying cell migration in naturally occurring environments, such as metastasising cancer in cell tissue or stem cells during embryonic development, proves immensely complex from an experimental point of view. Additionally, the external cues of the complex environment makes it difficult to develop a consistent mathematical description of the cell trajectories. To simplify the system, we constrain single cells to a quasi-one-dimensional migration by using micropatterned substrates that only allow cell movement along a fibronectin coated lane. The defined geometry facilitates quantitative read-out of locomotion, allows for large number statistics and simplifies theoretical models. In order to capture all relevant timescales of cell migration we observe cells for several days with a time-resolution in the order of seconds. From this we extract the memory kernel for a generalised Langevin equation approach. By comparing memory kernels, we are able to discriminate between different cell lines. In the next step we are interested in how external cues influence the characteristic memory kernels of cells. In particular, chemical gradients can be studied to get a better understanding of the cell migration process and its dependence on the microenvironment.

BP 18.21 Tue 14:00 P2/2OG

**Quantitative Phase Imaging for Studying Cell Migration and Bone Resorption in Periodontitis** — ●FELIX PETER SANCHEZ KLOSE, AGNES DAHLSTRAND RUDIN, KARIN CHRISTENSON, MARIA RANSJÖ, and JOHAN BYLUND — Göteborgs Universitet, Göteborg, Sweden

Periodontitis is a chronic inflammation of the tooth supporting structures caused by a prolonged immune reaction to bacteria resulting in tooth loss if left untreated. As an inflammatory disease, the migration of neutrophils to the site of infections is of interest to study periodontitis. Besides the inflammatory lesions, periodontitis is characterized by loss of the bone structures surrounding the teeth. Imaging of the cell cultures was performed with a quantitative phase imaging (QPI) microscope. We demonstrate that a QPI microscope can be used to measure neutrophil chemotaxis and to perform cell tracking on a cellular level. The system allows for statistical analysis of various chemotactic parameters. In an osteoclast model system, the live imaging allowed for identification of early osteoclast-like cell formation and helped us to pinpoint the ideal culture length for cell quantification. QPI offers a valuable tool to observe neutrophil migration in a label-free system directly in cell cultures. In contrast to conventional methods, QPI can give insights in the neutrophil migration on a single-cell level and it enables analysis of cell movement parameters, e.g., motility. In addition, this method can aid in investigating the processes of cell differentiation, e.g., formation of osteoclasts.

BP 18.22 Tue 14:00 P2/2OG

**Engineering motile amoeboid cells toward precise and targeted microtransport** — ●SETAREH SHARIFI PANAH<sup>1</sup>, OLIVER NAGEL<sup>1</sup>, VALENTINO LEPRO<sup>1,2</sup>, ROBERT GROSSMANN<sup>1</sup>, and CARSTEN BETA<sup>1</sup> — <sup>1</sup>Institute of Physics and Astronomy, University of Potsdam, 14476 Potsdam, Germany — <sup>2</sup>Max Planck Institute of Colloids

and Interfaces, 14476 Potsdam, Germany

Over the past decades, growing efforts have been invested on bio-hybrid microsystems. However, applications such as targeted drug delivery through complex environments remain challenging. Inspired by leukocytes, we propose to exploit the potential of eukaryotic cells for microtransport, using the *Dictyostelium discoideum* cells as model. Our experiments highlight the ability of amoeboid cells to displace cargo particles with faster spreading cargo-loaded cells, suggesting cell-particle interactions as a stimulus promoting cell motility. Further, we developed a physical model which captures the main motility aspects of our system. Chemotaxis experiments reveal a guided transport driven by both single and collective cells. Remarkably, active microtransport across 3D collagen matrices suggest reliability of our system also in complex environments. To get an insight into the cell-particle dynamics, the approximate pulling force exerted on a cargo by the cell, is being estimated using microfluidics. Interestingly, under constant drag force of up to a few nano Newton, agent cells maintain their adhesion site to the cargo while being pulled downstream, suggesting limited yet considerable applied force to the cargo. These findings serve as a basis for understanding underlying mechanics of such microtransport.

BP 18.23 Tue 14:00 P2/2OG

**Physical interaction of tumor spheroids with the ECM** — ●ELIANE BLAUTH, STEFFEN GROSSER, FRANK SAUER, and JOSEF A. KÄS — Soft Matter Physics Division, Peter Debye Institute for Soft Matter Physics, University of Leipzig, Germany

The interaction between tumors and the extracellular matrix is an important factor during cancer progression. Most experimental assays that are used to study this interaction are rather designed with single cells or have a complex three-dimensional structure.

Here, we present a simple, but efficient setup to characterize how model tumors interact with their surroundings. To mimic the physiological conditions in a more realistic manner we use three-dimensional tumor spheroids and put them on collagen matrices to model the extracellular matrix. With this approach our ECM-model gains a third dimension and complex interactions such as three-dimensional cell migration or the nonlinear viscoelastic behavior of the ECM are observed. By using multicellular spheroids as model tumors we detect not only the interaction of cell clusters with the ECM but also the different migration modes of cells that left the original spheroid. Still we can use basic brightfield microscopy to measure all of these properties sufficiently.

Our data demonstrate the invasiveness of different cell lines, including healthy epithelial ones, which is characterized by the traction forces and the collective migration behavior of the spheroids on different collagen matrices.

BP 18.24 Tue 14:00 P2/2OG

**Bacterial adhesion and biofilm inhibition on nanopatterned surfaces** — ●CLAUDIA ARBEITMAN<sup>1,2</sup>, MAGALÍ LINGENFELDER<sup>3</sup>, KARLA BANJAC<sup>3</sup>, PABLO ROJAS<sup>2</sup>, VIRGINIA ALBARRACÍN<sup>1</sup>, and MARTÍN GARCÍA<sup>2</sup> — <sup>1</sup>CONICET, Argentina — <sup>2</sup>Theoretical Physics, University of Kassel, Germany — <sup>3</sup>Max Planck - EPFL, Switzerland

The ability of the bacteria to associate into communities in biofilms -as it happen in medical devices- is central to their pathogenicity as they confer protection from antimicrobial agents and bactericidal molecules present on host tissues. Here, we developed a hierarchical antibacterial nanopatterning, that is able to control bacterial biofilm formation. We successfully tested this nanopatterned surface against multi resistant bacteria, proving a long standing protection with improved biocompatibility. In order to unveil the underlying mechanisms, we applied a set of advanced microscopy tools and mathematical models. Our results indicate further details in the realm of bacteria-surface interactions.

BP 18.25 Tue 14:00 P2/2OG

**Active gel model of onset of the movement in one-dimensional cell motility** — ●YUAN-HENG TSENG<sup>1</sup> and HSUAN-YI CHEN<sup>1,2</sup> — <sup>1</sup>Department of Physics, National Central University, Zhongli, Taiwan — <sup>2</sup>Institute of Physics, Academia Sinica, Nankang, Taiwan

To understand how the intracellular mechanical mechanism affects the motion of a cell, we develop a one-dimensional model for cell migration on a solid substrate. This model includes contractile force from actomyosin network, viscous stress in the cytoskeleton, actin polymerization at the ends of the cell, drag force due to substrate and drag force due to cell-substrate bonds. Our numerical solutions show that in addition to the contractility, polymerization-cell-movement feedback, and

spontaneous symmetry breaking in the distribution of cell-substrate bonds helps to initiate cell crawling. This is illustrated by phase diagrams for static and motile states.

BP 18.26 Tue 14:00 P2/2OG

**Motility of aggregated *Neisseria gonorrhoeae* cells influences cell death and response to antibiotic treatment** — ●MARC HENNES, TOM CRONENBERG, and BERENIKE MAIER — Institute for Biological Physics, AG Maier, University of Cologne, Germany

Like most procaryotic unicellular organisms, members of the genus *Neisseriaceae* polymerize hair-like appendages that protrude from the cell body. In the case of *N. gonorrhoeae*, the pathogen responsible for the sexually transmitted disease gonorrhoea, the so-called type IV pili (T4P) allow the cell to bind to surfaces and move on them through retraction, but also enable the uptake of extracellular DNA. Cooperatively, intercellular network binding by dynamic T4P-T4P interactions leads to the formation of spherically shaped cell aggregates which may fuse and deform like water drops and play a crucial role in the infection of host tissues. Here, we investigate the temporal dynamics of these aggregates at the single-cell level by tracking the positions and velocities of each bacteria. We find that bacterial motility inside the colonies is highly heterogeneous and sensitive to translation inhibitors, which

hyper-motilize bacteria and homogenize their dynamics. Strikingly, we find that cell motility correlates negatively with the fraction of dead cells in the absence of bactericidal antibiotics, and positively in their presence.

BP 18.27 Tue 14:00 P2/2OG

**A large scan are AFM with convolutional neural network image segmentation for live cell measurements** — ●TODOR KRASSTEV and TILMAN E. SCHÄFFER — Universität Tübingen, Institut für Angewandte Physik, Auf der Morgenstelle 10, 72076 Tübingen

The atomic force microscope (AFM) has become a robust and versatile tool for the investigation of mechanical properties of single cells. We have developed an AFM with a large scan range of 25 mm in xy-direction. This enables us to access high numbers of cells for mechanical measurements. In order to harness the full potential of our setup we employ state of the art convolutional neural networks for single cell detection, thereby facilitating high throughput measurements on single cells. To demonstrate the setup's potential we investigate the influence of common live fluorescent dyes on single cell mechanics. Our investigation is focused on SiR-actin and SiR-tubulin as these dyes target principal components of the cytoskeleton.